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SOME EXPERIENCES WITH CASES OF DRIVING UNDER THE INFLUENCE OF INTOXICATING LIQUOR IN VICTORIA: AN ESSAY ON DRUNK DRIVING.

By J. H. W. BIRRELL, M.B., B.S.,
Police Surgeon, Melbourne.

B.B.: But is he to be allowed to defy the criminal law of the land?

Sir Patrick: The criminal law is no use to decent people. It only helps blackguards to blackmail their families. What are we family doctors doing half our time but conspiring with the family solicitor to keep some rascal out of jail and some family out of disgrace? GEORGE BERNARD SHAW—"The Doctor's Dilemma", Act 3.

As the incredible total of road casualties increases day by day, a fact which has been obvious for many years to those intimately concerned with road accidents—namely, that alcohol is directly responsible for much of the carnage—is slowly becoming obvious to some other members of the community.

Together with other motorized communities, Victoria has legislation which, in theory, is designed to cope with the problem of alcohol on the roads. However, it is becoming rapidly apparent, that the legislation is based on a presumption, which, to say the least, is doubtful—that is, the driver must be obviously affected by liquor and, in effect, in that popular state known as drunk before he can be considered a danger on Victoria's roads. This is disturbing, but is on a par with the assumptions on which many of our road laws are based. Few, if any, of these assumptions have been put to the usual rigorous tests of scientific proof which one is accustomed to use in other fields which materially affect the community's welfare.

It is proposed in this paper to state the law in Victoria, examine the affected drivers and then see how the law treats them in practice.

The Law.

The Victorian legislation dealing with the offence of "driving under the influence of intoxicating liquor" is to be found in the Victorian *Crimes Act* 1958, Sections 319, 320, 321 and 408.

Section 319, sub-section (1), is in the following terms:

> Any person who drives a motor car under the influence of intoxicating liquor, or of any drug, to such an extent as to be incapable of having proper control of a motor car shall be guilty of a misdemeanour and shall be liable to be imprisoned for a term of not more than two years or to a fine of not more than one hundred pounds or to both such imprisonment and fine, and upon any conviction for the said misdemeanour the court shall if the offender holds any licence under the Motor Car Act 1958, cancel that licence and shall whether or not he holds any such licence disqualify

> him from obtaining any such licence except upon the order of a court of petty sessions consisting of a stipendiary magistrate sitting alone; . . . no such order shall be made to take effect before the expiration of a period of twelve months after the date of conviction.

Drug means any narcotic or dangerous drug within the meaning of the *Poisons Act* 1958. It does not include insulin, bromides or the bromureides.

Section 321 allows a Court of Petty Sessions to hear and determine the charge in summary manner, the person charged having the right to be tried by a jury. Penalties in Petty Sessions are a fine of not more than one hundred pounds or a term of imprisonment of not more than six months. In the case of a second or any subsequent offence, a penalty is provided for imprisonment for not more than twelve months.

Section 408 provides for the production of certificates (i) by the medical practitioner as to the taking of a sample of blood from the accused person; (ii) by an approved analyst as to the level of alcohol in grammes per 100 ml. of blood.

The section also provides that

> . . . where any legally qualified medical practitioner is requested to make any examination or to collect a sample of blood . . . and where the person to be examined or from whom a sample of blood is to be collected has expressed consent to such examination or collection no action shall lie against the legally qualified medical practitioner who, notwithstanding that it subsequently appears that the person was in fact incapable by reason of his mental condition from effectively giving consent to the examination or collection.

Further, "the mere failure or refusal to express consent shall not be used in evidence against or referred to in any way against his interests in any proceedings".

Sub-section (2) of section 408 provides that if the analyst's opinion is that the percentage of alcohol was 0·05 or less at the time of the alleged offence, that opinion shall be taken as prima-facie evidence that such person was not under the influence of intoxicating liquor to such an extent as to be incapable of having proper control of a motor-car at that time. There is no status for a blood alcohol level above 0·05%; the level may be accepted with other relevant and admissible evidence.

Regulations have been gazetted governing the taking of blood samples. Apart from obvious matters such as no alcohol being used in the preparation of the arm or the syringe, it is stated expressly that the tubes used for collecting the blood shall contain 15 mg. of potassium oxalate and 5 mg. of sodium fluoride as anticoagulant and yeast inhibitor respectively.

Under the *Motor Car Act* of 1958, section 82, a person is guilty of an offence if under the influence of intoxicating liquor while in charge of a motor car to such an extent as to be incapable of having proper control. The penalties are not as severe as the driving offence, and the driving licence is not automatically cancelled. However, the person is not "deemed to be in charge of a motor car unless he is attempting to start or drive the motor car".

The Drivers.

The material on which this paper is based includes some 1009 drivers personally examined by the writer at the request of Victoria Police over a period of 27 months from August, 1957. These 1009 drivers were almost all charged by police with the offence of driving under the influence of intoxicating liquor. Over this same period, some thousands of other drivers were also examined whilst under the influence of liquor but, in the opinion of police and/or doctor, were not, under the present law, sufficiently and demonstrably affected to be charged with the offence. Many other drivers in all stages of intoxication were admitted straight to the hospital or to the morgue.

The drivers were all apprehended by police in Melbourne and its suburbs, no country drivers being included. Most drivers were either examined and/or had blood samples taken. These 1009 drivers were responsible for 50 road deaths and many injuries. In the same 27-month period, 2800 drivers were charged with driving under the influence of intoxicating liquor in the whole of Victoria. The total dead and injured on Victorian roads over this period were 1304 killed and 33,049 injured.

The Patterns of Alcoholic Collisions.

Clearly any and all driving faults in the book may flow from the combination of drinking and driving when the drinking is more than very moderate. In the group of drivers considered in this paper, the drinking was in every case more than moderate.

Only 30% of these drivers arrested for driving under the influence are actually found driving the vehicle. The certain reason for this is that police are so busy attending collisions that there is little time for observation of driving. Police are also dangerously short-handed, there being one traffic policeman for every 615 miles of highway and main road in Victoria. Suffice it so say that 70% of drivers arrested for driving under the influence have been involved in a collision.

Collisions with stationary objects are almost synonymous with a driver who is well under the influence of alcohol or who has taken alcohol while other factors are operating, such as fatigue (Haddon and Bradess, 1959; Birrell, 1960). The driver who is grossly affected by alcohol has difficulty in controlling his car at all; if he is able to get the car moving, he is completely oblivious of other vehicles or objects on the road. Sometimes he may be so affected that he is unable to start the car. One man recently was so much under the influence he overbalanced putting his foot on the self-starter and fell out of the car.

If this type of driver gets his car going he will often collide at slow to moderate speed with an adjacent tree, parked car, pole, post or safety zone. The number of flashing lights on the zone appears to make little difference. Another common place to find this driver is crashed into the back of a car stopped at a railway crossing, red light or waiting correctly to do a right-hand turn.

Occasionally this driver gets up some speed and kills.

On more open roads there are several patterns which involve speed; here, of course, driver depreciation due to alcohol, which may not be particularly dangerous at low speeds (more tracking movements with the steering wheel than is normal is the typical sign), may become very dangerous at high speeds. Often on straight roads particularly, in the early hours of the morning, alcoholic drowsiness takes over at speed, the car overturning or colliding with a stationary object. This is the pattern for so-called road suicide, when a lone driver kills himself.

The worst collisions occur on our best and widest roads, the driver under the influence veering at speed to the incorrect side of the road—a truly frightening and only too common situation on Victorian roads.

The victim is either hit before he can make a move or is hit trying to go round the other side of the offending driver. This last was the pattern for one of Victoria's worst accidents, in which six men in a car collided with a transport vehicle. The lowest blood alcohol level of the six men in the car was 0·17%; they were all killed.

Occasionally the victim crashes while taking evasive action, the offending driver under the influence continuing on his merry way.

Another unfortunately common and indeed despicable pattern for the alcohol-affected driver is the hit-and-run accident. However, occasionally the hit-and-run offender is so drunk that he does not even know he has had an accident.

The Drunk Driver.

Age.

The average age of the driver in this series is 37 years (Figure I). This age group is considerably over-represented compared with its proportion within the community.

It is an interesting point that the killer driver—that is, one who survives a fatal collison—tends to be below 30 years of age and to have a blood alcohol level much lower than the average level for a case of driving under the influence.

Sex.

The sex distribution is heavily loaded on the male side—that is, 995 men to 14 women. In effect, a little over 1% of drivers charged with driving under the influence are female. No figures are available as to the amount of driving done by women generally, but this proportion is obviously far below what would be expected if females combined drinking and driving as much as males. Indeed, it is seldom that one sees a female driver involved in a severe collision. Three of the females in this series were chronic alcoholics and another two practising prostitutes. Most were involved in some type of domestic trouble. Most were middle-aged and all very much drunk.

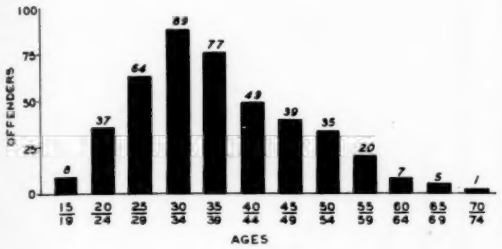

FIGURE I.

Ages of a group of drunk drivers arrested by Victorian police. The mean age is 37 years.

Occupations.

The occupations where known of 921 drivers are listed. Labourers, machinists and process workers numbered 194; professional drivers, 143; plumbers, fitters and tradesmen, 88; salesmen and sales managers, 79; managers, company directors and self-employed people, 71; clerks, 43; mechanics, 41; taxi drivers, 32; carpenters, 31; painters, 27; engineers, 20; contractors, 18; women drivers, 14; butchers, 14; bricklayers, 13; liquor trade employees, 12; soldiers, 10; grocers, 8; foremen, 8; accountants, 7; doctors, 7; policemen, 7; farmers, 6; chemists, 5; draughtsmen, 4; architects, 3; dentists, 3; firemen, 3; journalists, 3; teachers, 3; lawyers, 2; blacksmith, 1; driving school instructor, 1.

The striking feature is the large number of drivers who depend on driving for their living or whose occupation would be impossible to carry on without a driving licence. The number of taxi drivers is rather terrifying, particularly as several were carrying passengers at the time they were arrested! The range and extent of the occupations listed indicates that all sections of the community drink to excess before driving, few, if any, groups being immune from the disease of drunk driving. Too many of the so-called upper classes are included, but amongst these are a number of concealed chronic alcoholics.

Drinking Experience.

An instructive feature and one which the community could well take to heart is that at least 60% of people driving under the influence claim to be good, practising, experienced drinkers. So they should be, because many of them claim to have had 10 or more beers every night or two pints of whisky a day. "I've always got blood in my alcohol, doctor", one executive-type alcoholic told me.

Many of these good drinkers come from occupations such as those of salesman or advertising executive, in which heavy social drinking is said to be indispensable to the carrying on of the occupation. Clearly alcohol must add considerably to the overhead costs of business generally.

Only two drivers were drinking for the first time, one having the surprisingly high blood alcohol level of 0·18%.

Epidemiology.

The epidemiology of Victoria's drunk driving appears largely dependent on hotel closing time at 6 p.m. and closely parallels road accidents and casualties (Birrell, 1960). Most drunk drivers are found between 6 p.m. and 8 p.m. on Friday and Saturday nights; these hours account for 56% of the total number per week. It is interesting to compare Victoria's 6 to 8 p.m. peak with the 10 p.m. to midnight peak found in Queensland and England, where hotels close at 10 p.m. (Hayes, 1961; Birrell, 1960).

Drinking Habits.

A number of chronic alcoholics are represented—that is, people who cannot stop drinking. Usually they have enlarged livers and some peripheral neuritis. Many of these are still driving, the evidence being insufficient to convince the bench and particularly the juries. However, most are simply heavy drinkers. How many will go on in time to true chronic alcoholism is impossible to predict. However, the number of true alcohol abusers would be less than has been found, for example, in Sweden (Goldberg, 1955). The reason for this is the completely different approach in Sweden, where the ordinary drinker has been taught to leave his car behind. In Victoria, everyone drinks and drives, there being neither example nor, as will be seen later, practical deterrent.

Some of the drivers in this series are lone drinkers, but the majority appear victims of the drinking school with its rigid customs of paying for equal numbers of drinks and "not dragging one's chain"—that is, keeping up with the fastest drinker.

Type of Beverage.

Ninety *per centum* of the drivers charged with driving under the influence have been drinking Victorian beer and beer alone. Indeed, many appear almost insulted when it is suggested that they could have been drinking spirits. The average Victorian beer is strong, containing 5% of alcohol by volume. It is also most palatable, and is said to be one of the best in the world. However, it would appear that there is great need of a lighter brew, because the individual drivers appear to consume enormous quantities.

A proportion of drivers (in the region of 30%) give a history of worry, domestic and/or financial, which they usually blame for their excessive drinking.

Drug Association.

In seven cases, alcohol has been associated with a drug. Three drivers (two truck drivers and one taxi driver) had high blood alcohol levels (above 0·2%) associated with "Benzedrine" (amphetamine sulphate). Two drivers had little alcohol with an overdose of barbiturates, and the other two had little alcohol associated with an overdose of bromureide compounds. The "Benzedrine" victims were all very violent and belligerent. Many chronic alcoholics use a little alcohol with the readily available bromureide compounds to produce a financially cheap state of intoxication. It is suspected that "Benzedrine" usage is extremely common amongst transport drivers, but lack of adequate investigation and supervision prevents any decision on this point.

The Blood Alcohol Level.

Figure II illustrates the range of blood alcohol levels in 611 drivers, the great majority of whom were charged with driving under the influence of intoxicating liquor. Only two of those with levels under 0·1% were charged with the offence, the remainder being either under the influence of drugs or involved in a fatal collision. It can easily be seen that the great majority of drivers charged with driving under the influence of intoxicating liquor have a blood alcohol level well in excess of 0·15%, the average being 0·215%. The dearth of cases of levels between 0·05% and 0·15% is explained by the fact that police arrest only those drivers showing very obvious signs of alcoholic intoxication. The law takes little or

no notice of the accident-producing blood levels 0·05% to 0·15%. Indeed frequently it takes no notice of blood levels considerably in excess of 015%.

The higher levels—that is, those above 0·25%—take some explaining since they represent relatively enormous amounts of liquor. In many cases, such levels represent all-day drinking following a heavy drinking session the previous night. "I'm shober now, but I was very drunk last night" is a common story.

the lower blood levels. However, the test is quite accurate enough to be used in an analogous manner to the speed limit—that is, as a drinking limit.

The Law in Practice.

In practice legal argument in Victoria is directed to how drunk or how much under the influence of liquor one can be before one is considered incapable of having proper control of a car. There appears no harder task

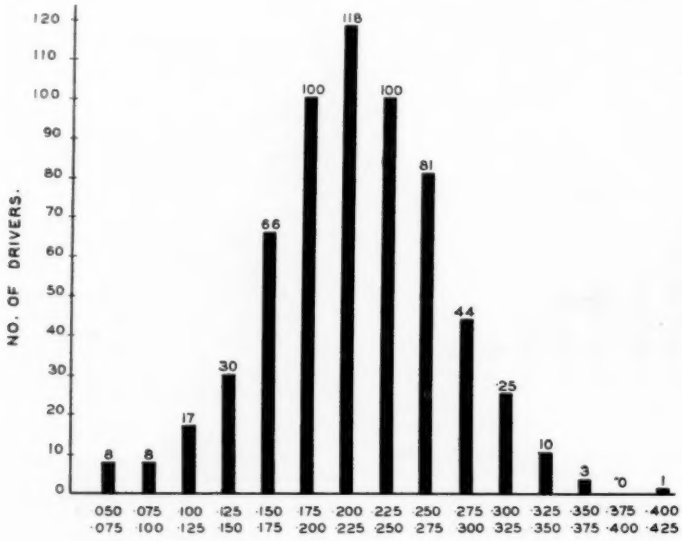

FIGURE II.
Blood alcohol levels in 611 drivers.

Most importantly, some 10 drivers have had their innocence conclusivey proved by the demonstration of negligible blood alcohol levels, at least in so far as alcoholic intoxication is concerned. These cases, unfortunately, do not make news, but illustrate the great value of the test as a test of innocence.

The 10 drivers included a patient with Huntington's chorea, a sufferer from sunstroke who had had two beers, a man with a hypoglycæmic coma, a senile dement, an epileptic, two patients with barbiturate intoxication and a driver who was thought from his driving to be drunk.

Early in this work about 30% of drivers volunteered for the blood alcohol level test, but acceptances have dropped to a minimum; those who accept comprise a few badly-intoxicated people and some braggards, all of whom have lost count after six beers—as do most drinkers.

The percentage of acceptances has particularly dropped after unfavourable publicity being accorded to preventive aspects of drunk driving by various organizations. The evening Press has had headlines and even leaders proclaiming the blood alcohol test as inaccurate. This is somewhat of a libel on the analytical chemists since the test in experienced hands is one of the most accurate biological tests we have. It is apparent that one particular journal has been unable to distinguish between the living and the dead, because although there is some argument as to the significance of exact post-mortem alcohol levels, there is no argument as to the reliability of the alcohol level obtained from venous blood in the arm in the living body. This level is an accurate index of the total amount circulating in the body and also of the minimum quantity of liquor required to reach that particular level.

Naturally, as with ordinary biological variation, it is impossible to pin-point accurately an individual's degree of intoxication at a given blood level, particularly at

in trial procedure for the prosecution. Indeed, with money and a capable barrister there should be no convictions because of the possibilities which can be raised in defence of a charge of driving under the influence of intoxicating liquor.

Proofs required by the courts on the part of the prosecution are two. Firstly, it must be proved that the defendant was actually driving the motor car. It would appear that the standard of proof of driving is extraordinarily high.

A man drove his small car on a wide, four-lane highway in broad daylight into the back of a large, very visible 'bus. He was found sitting behind the wheel seconds later by the bus driver and his conductor. He admitted to the bus driver, to police and to the doctor that it was his car, that he was driving it and that he was drunk and not injured. His blood alcohol level was 0·268%. The case was dismissed because there was no evidence of driving.

Secondly, it must be shown that at the time of such driving he was under the influence of intoxicating liquor or drugs to such an extent as to be incapable of having proper control. Theoretically, it is possible for a charge to be proved by evidence of the defendant's condition of sobriety, together with an expression of opinion by either police or a medical officer that he was incapable of having proper control of a motor car. However, in practice courts require more evidence to be satisfied that even a drunk person was incapable of having proper control. In the absence of evidence of abnormal driving, courts are apt to be not satisfied beyond reasonable doubt of the fact that the defendant was incapable of having proper control. We therefore frequently have the spectacle of the man who was drunk escaping any penalty because of the absence of evidence of bad driving at the moment of apprehension.

It is at this point that both the medical examination for sobriety and the blood alcohol level test, under the present

law, leave much to be desired. Both naturally are usually done some time after the event, although generally within two hours. Obviously both do not give the results which they would if done at the time of driving. However, even assuming that both sobriety test and blood test are admitted as evidence, frequently they have little effect, as the following case illustrates.

It was argued in the case of an appeal by a young man, aged 21 years, against a conviction for driving under the influence, that if he could add up a sum of money, add mentally 6, 12 and 5, and write his name and address "not badly", then this indicated that his higher cerebral functions were in order and that he was fit to drive. It was added that it did not matter about his slurred speech, staggering gait, poor balance and blood alcohol level of 0·18%, since he did not need these to drive a car. The actual driving in this case (45 miles per hour in a 30 miles per hour zone, cutting in and out of traffic, just missing cars and kerbs over a mile or so) was considered not dangerous but clever driving. The appeal against the conviction was allowed.

This is doubtless a strict literal interpretation of the law, but does indicate just how far from reality the law in this matter has receded.

Clearly the law, apart from being based on the assumption that the drunk driver is the dangerous driver, takes no account of potentiality. For example, an almost paralytically drunk man who is seen to drive only a few hundred yards in a straight line, and is stopped before he does any harm, will not usually be convicted.

The frequent concurrence of injury and drunkenness is taken full advantage of by the defence, the physical signs of staggering, slurred speech and so on being due always to "concussion, nervous shock or cerebral commotion". It is always conveniently forgotten that shock or concussion does not explain the driving before the accident nor, to my knowledge, does shock or concussion put alcohol in the blood-stream of those few people who volunteer for a blood alcohol test. It is also forgotten that alcohol in any quantity is a magnificent drug for stopping worry and nervous shock, and is also analgesic—witness one's own bruises the morning after.

TABLE I.

Conviction Rate in English County Compared with Victorian Rates.[1]

Year.	Number Electing to be Tried Summarily by Magistrate.	Percentage of Convictions.	Number Electing to Go for Trial.	Percentage of Convictions.
English county:				
1953	115	92	8	38
1954	161	94	20	35
1955	166	92	13	46
1956	197	89	30	33
1957	197	91	37	35
Victoria:				
1956	1051	79	26	10[2]
1957	1178	67	34	10[2]
1958	1375	78	99	24[2]

[1] Figures for English county taken from Cohen *et alii* (1958).

[2] The majority of convictions were for the alternative charge of dangerous driving, the drunk driving being dismissed.

A lower conviction rate before the jury is found for the crime of manslaughter with a motor-car; most cases involve alcohol.

The conviction rates are interesting, particularly the rate in petty sessions as opposed to the rate before the jury in general sessions. These are compared with the figures of an English county (Cohen *et alii*, 1958) (Table I). There is no apparent difference in the degree of severity of cases going for trial compared with those staying in petty sessions. People disliking publicity tend to stay in petty sessions whilst those with money and/or prior convictions frequently elect to go for trial.

The reasons for the dearth of convictions before the jury are manifold, but one feels that there is no standard of measurement which the jury can use as a guide. There is also the only too common attitude of: "There, but for the grace of God, go I." In effect, the only stigma of a drunk driving conviction in Victoria is the inference that the driver is a poor drinker.

Discussion.

It is curious to realize that there is no definition or standard of the word "drunk" or of being under the influence of intoxicating liquor to such an extent as to be incapable of having proper control of a motor-car.

Three examples may illustrate the difficulty.

A young man, aged 27 years, was driving a three-ton truck without headlights in the fourth lane on the wrong side of a four-lane highway. (Even four-lane highways are not wide enough for many drivers under the influence of liquor.) He had great difficulty in articulation and in standing upright, and had a blood alcohol level of 0·25%. He stood on his hands several times, a feat which neither the doctor nor police could do sober.

An ex-policeman, aged 36 years, hit two consecutive lamp posts on the wrong side of the road; instead of levering his dented fender off the wheel, he was about to crash a third post in an attempt to remove the offending fender when police apprehended him. He was unable to stand upright without support and his speech was unintelligible, yet when asked, he walked the line quite well and was even able to stand on one leg. He refused a blood test.

An accountant, aged 40 years, was colliding with the kerb on alternate sides of the road. He vomited shortly before being stopped by police. He had to be assisted from the car and held upright. His writing and printing, done while seated, was very good. He also refused a blood test.

In each of these three cases the driver was in the state popularly known as drunk, yet each man was able to perform part or most of a so-called sobriety test in a fairly creditable manner—at least while he concentrated. It is of interest that only the first man was convicted. It is apparent, therefore, that the routine clinical examination has a considerable number of gaps, even when use is made of a battery of so-called coordination tests. Most of the tests used are simple and straightforward, and can be done easily by good drinkers even at the stage when they can "hardly scratch themselves".

This can be confirmed any night on Victoria's roads. For example, police attention is attracted to a particular car. They chase it with siren blaring and lights flashing; then they pull alongside shouting at the driver to stop. He, well under the influence of liquor, has neither eyes nor ears for anyone or anything else on the road, being intent simply on keeping his car somewhere on the road. When his attention is attracted, the car wavers all over the road and, as often as not, nearly rams the police.

This is a phenomenon which one may call attentiveness, and in effect, it means that the man under the influence of liquor tends to attend to one factor only in a driving situation. This explains why particularly in the blood alcohol level ranges of 0·05% to 0·2%, giving drivers simple tests produces many results which are reasonably normal since, in fact, the driver, by virtue of the simplicity of the test, has been given all the opportunity in the world not to show any effects.

This phenomenon has shown up very well in recent work in both England (Drew *et alii*, 1958) and in Canada (Coldwell, 1957; Penner and Coldwell, 1958), where actual or simulated car driving has been used as a test medium. It was found that drivers who clinically were not obviously affected by liquor would watch the path of the car and forget signal lights or watch the lights and forget to steer.

Blood samples are infrequently taken and even less frequently are the results of blood tests admitted as evidence. When admitted, the lack of guidance as to the meaning of a given level, either as a simple level or in terms of the minimum amount of liquor consumed, results in courts disregarding the blood alcohol level as evidence.

However, when admitted, even under present circumstances, the blood alcohol level does very speedily get rid of the usual defence of no drinks or few drinks.

A driver was followed for several miles meandering gently over suburban roads. Eventually he was stopped by police. He was frankly and admittedly drunk, saying that he had

had a few beers shortly before being apprehended. Then he refused to have a blood sample taken. He smelt strongly of intoxicating liquor. The Court accepted the evidence of one friend of the driver, who stated that the driver had not had one drink that day and night, but had had some salad with some vinegar in it. The case was dismissed.

The over-all picture which emerges is not a pretty one. Daily perjury (not always intentional) is accepted of the number of drinks taken; drivers well under the influence at the time of admission to hospital are suing and being sued as sober drivers; the police force is discouraged and disheartened, and a bewildered community is in ignorance of what really goes on. The classic illustration of confusion is the statement that the fatality rate has dropped from 17 to 12 per 100,000,000 miles travelled in the last 10 years. This is a world-wide phenomenon, largely owing in all probability to the efforts of the medical profession in keeping more people alive after bad collisions. But we ignore the fact that the over-all casualty rate in the same period has risen from 230 to 330 per 100,000,000 miles travelled in Australia.

Members of our profession, apart from those working with road accident victims, are not fully aware of the problem. No general practitioner can afford the time to examine more than an occasional police case, since each case is liable to result in the loss of a working day. Those who do examine an occasional drunk driver at the request of police become rapidly disillusioned at the unreality of the procedure.

When the practitioner can, he confines himself simply to taking the blood for an alcohol level estimation, leaving the police to produce evidence of drunkenness. This last must be very obvious before the courts take notice.

As a result of the medical practice of taking blood and avoiding any further involvement, the claims of anxiety states, blackouts and so on are very common defences and are mostly completely successful. It is astounding the number of anxiety states found amongst Victorian drivers.

Any medical man who points out the dangers of combining drinking and driving, or who insists that alcohol is an important factor in road accidents is immediately assailed in many arenas as a wowser, while his work is politely deprecated on the grounds of youth, inexperience and heaven knows what else. However, few of these assailants would drink from a source of water which that same medical man had declared contaminated by typhoid.

The community is uncomfortable about this sort of sociological problem—that is, it wants to and it does not want to do anything about the problem.

The familiar human way out of such predicaments is to attack the problem in such a way as to ensure minimum results (Moynihan, 1960).

As far as the legal situation is concerned, one eminent legal man put the matter in a delightfully concise fashion recently, when he said: "If you see a man come out of an hotel and stagger down the street, with slurred speech and disarranged clothing, then he is drunk. If that same man in the same condition gets behind the wheel of a motor-car, then the matter cannot be determined." Put in another way, only gentlemen drive motor-cars, and gentlemen do not get drunk or drive their motor-cars whilst under the influence of intoxicating liquor. Unfortunately, neither of these propositions has been found correct in practice.

Conclusions.

1. The medical examination and sobriety tests are not nearly accurate enough to assess alcoholic impairment in relation to driving.

2. The drivers charged with driving under the influence of intoxicating liquor in Victoria form only a small fraction of the total number of drivers actually driving under the influence of intoxicating liquor.

3. The laws relating to drunk driving are not geared to modern traffic, and have little relation to reality. They have also little deterrent effect, as is evidenced by the range of occupations of Victorian drunk drivers, a great proportion of whom are professional drivers or depend on their driving licences for their living. They are mostly experienced drinkers.

4. The chemical test for alcohol has little status, and is hamstrung as evidence by poorly drawn regulations. Despite this, it is an accurate and objective test—more accurate than most of our routine tests for biological fluids—whose value is perhaps best evidenced by the efforts being made in Parliament, in the evening Press and other places to discredit it.

Summary.

Drunk drivers and their treatment by Victorian law are examined. Their average age is 37 years, and there is a great predominance of males, while most have been drinking beer and are good drinkers. The average blood alcohol level is well above 0·2%; 70% are found because they have been concerned in collisions. All sections of the community drink and drive to excess.

The legal and medical position is difficult. It is concluded that the law has moved away from reality; those dealt with by the courts for this offence are a small proportion of culpable drivers seen under the influence of intoxicating liquor. The sobriety tests used, such as walking a line, are a ridiculously inefficient means of determining driving impairment in relation to liquor consumed.

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THE DRINKING DRIVER.

By G. S. HAYES, M.B., Ch.M.,
Government Medical Officer, Brisbane, Queensland.

IN Australia, as in other countries, the problem of the drinking driver is engaging the increasing attention of those concerned with the public safety and traffic control.

The fact that the term "drinking driver" is being used more frequently than the former "drunken driver" is an indication of an increasing awareness that a motorist—over-careless or unduly exuberant as the result of alcohol—is as great a menace as the incapable or completely befuddled drunk, and that he can be even more dangerous, for the latter usually does not get far, even indeed should he succeed in starting his vehicle.

With more congested traffic and higher road speeds, it is not a question of when a man is drunk, but rather when he begins to become careless or less attentive to possible danger. The extreme view is expressed in the lines:

> He is not drunk who from the floor
> Can rise, and drink once more:
> But he is drunk who prostrate lies,
> And can neither drink nor rise.

and also by the tale of the reveller staggering towards his car who, when asked by a policeman whether he intended to drive his car, replied: "Of corsh offisher — I'm too drunk to walk."

On the other hand there is a quite unwarranted fear that the intending motorist should not under any circumstance allow alcohol to pass his lips for several hours before driving. Any person accustomed to the moderate use of alcohol — still, if wisely used, the best of all the tranquillizers for stress and strain — should be able to have his couple of "noggins" or "snorts", with one for the road, without their impairing his normal skill or adversely affecting his mental or physical faculties. In other words — the normal, sensible man with some experience of alcoholic

FIG. I. SHOWING PERCENTAGE OF ARRESTED ALCOHOLIC DRIVERS IN BRISBANE (1952-59), OF DRIVERS WITH TRAFFIC ACCIDENTS (QUEENSLAND) 1952-59, & BRISBANE MALE POPULATION IN VARIOUS AGE GROUPS (1954 CENSUS).

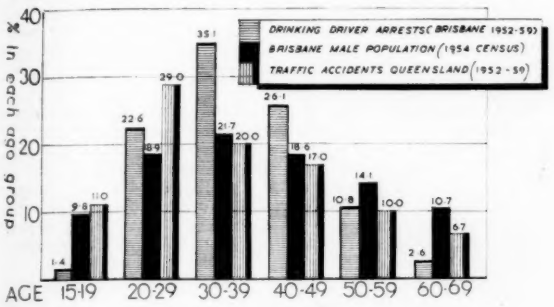

FIGURE I.

Graph showing percentages of arrested alcoholic drivers in Brisbane (1952-1959), of drivers with traffic accidents in Queensland (1952-1959) and the Brisbane male population in various age groups (1954 census).

beverages should be able to have his three beers or three whiskies after work without getting into trouble or becoming a traffic risk. Experience in Brisbane goes to show that practically all persons arrested on a drinking-and-driving charge have either been involved in a traffic accident to which their carelessness contributed, or have attracted police attention by their abnormal behaviour and have had considerably more. It is partly my purpose in writing to show that the moderate, sensible drinker does not as a rule get involved in drinking-and-driving charges, although, on the other hand, a few more drinks than usual may land him in trouble long before he reaches the "publicly drunk" stage.

During the years 1952-1959 inclusive, 2645 drinking drivers have been medically examined at the City Watchhouse in Brisbane — 1744 by myself and 901 by seven colleagues acting at different times as relieving Government medical officers, and the experience and records collected from these examinations, and in many cases from subsequent court proceedings, form the basis of this report. If the year 1960 is included with this period, 3222 examinations were carried out, but the extra 568 cases for 1960 do not materially alter the general picture, and so this article is based on the 2645 consecutive cases for 1952-1959 (Figure V).

However, firstly I want to summarize the law in Queensland, particularly as it applies to Brisbane — a growing metropolis of over half a million people. All Australian States have legislation making it an offence for anyone to drive a motor vehicle whilst under the influence of alcohol. The *Queensland Traffic Act* of 1949-1959 is typical, although perhaps a little more sweeping than those in other States. Section 16 of this Act states that "any person who whilst under the influence of liquor or a drug drives, or is in charge of, a motor vehicle, or who occupies the driving seat and attempts to put the vehicle in motion shall be guilty of an offence". As the term "under the influence" is not further defined, and as "being in charge of" does not necessarily imply being either inside or even alongside the car, there was naturally some apprehension that the rather sweeping terms of the Queensland Act might result in unwarranted arrests by over-zealous policemen. A respectable citizen after one or two whiskies would have the odour of liquor on his breath, and if involved in a minor traffic incident, through no fault of his own, could be charged under this section, as even one whisky does have some effect physiologically, albeit not necessarily adverse.

Since the implementation of the Act in 1951 the police have guarded against the possibility of such abuses by

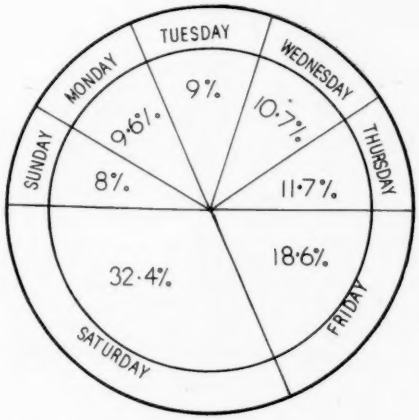

FIGURE II.

Diagram showing the days of the week on which the drinking drivers were examined.

having every person so charged examined by the Government medical officer for the district. It might be pointed out that in Queensland the Government medical officer is called upon to act as the police surgeon, but he is nevertheless an independent appointee not in the employ of the Police Department, and he is usually the senior medical practitioner in the district, acceptable alike to the general public on the one hand and to the Government on the other — thus his impartiality is ensured, and acknowledgement is made of the rule that not only should justice be done but it must also appear to be done. Additionally, police officers are warned against over-precipitate action, and the Policeman's Manual (paragraph 1557 (1) (b)) states: "Every member of the Police Force should realise the extreme importance of refraining from precipitate action against persons whom he suspects to be under the influence of liquor or of a drug, and take action only in those cases where there is specific evidence that the relevant offence has been committed."

Paragraph 1557(3) goes on to say:

> In all cases of arrest for offences against the provsions of section 16 of "The Traffic Acts, 1949 to 1957", the person arrested must be examined by the Government Medical Officer with the least possible delay following arrest. Members of the Police Force are also directed that where a person arrested for this class of offence requires his own medical adviser to examine him, his request must be complied with, in so far as either giving him facilities to telephone his own medical adviser, or the senior member of the Police Force doing so on his behalf, but whether or not there is a request for and/or an examination by a private medical practitioner at the instance of a prisoner, the police are not relieved from the obligation of immediate action to have an arrested person medically examined by the Government Medical Officer where such latter officer is available.

In so far as Brisbane is concerned, facts to be given later in this article will show that all the drinking drivers arrested in Brisbane in the past eight years have been examined by a Government medical officer, and that in 90·8% of these cases chemical tests have been performed, with results completely exonerating the police from the charge of precipitate action or unjustified arrest. The term D.I.C. (drunk in charge) has become the convenient appellation for these cases in Queensland.

In the greater Brisbane area if a driver is arrested he is taken to the City Watchhouse, where the Government

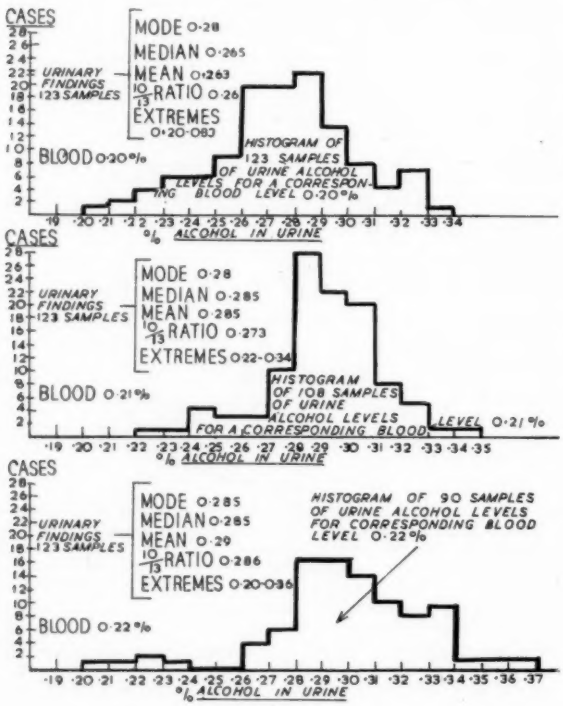

FIGURE III.

Histograms showing urine alcohol levels corresponding to blood alcohol levels of 0·20%, 0·21% and 0·22%.

medical officer is asked to examine him. This examination is carried out in a specially designed surgery separate from the main body of the Watchhouse, and no police are present. In most cases the medical examination is carried out within an hour of the arrest. The usual questions are asked and observations are made, but no set *questionnaire* is followed. I believe that not only do different people react differently to alcohol, but that each doctor has his own particular way of conducting his examination; however, the general lines follow those set out by the British Medical Association (1958). When possible, a urine sample is obtained and examined for sugar and albumin and a small amount (1 oz. is sufficient) of this is placed in a screw-top glass jar for later alcohol estimation. It is then suggested to the accused person that he should have a blood sample submitted to an analyst, and to this a big majority readily agree. A few refuse to have blood tests, and some are so obviously drunk as to be difficult subjects for venepuncture; but of the 2645 persons examined in this series 2196 (or 83%) had blood tests, and, including those whose urine was examined in the event of blood not being taken for various reasons, 2409 (or 90·8%) of arrested drivers had chemical tests for alcohol performed. Blood and urine specimens were collected simultaneously from 1700 persons and the ratio of blood levels to urine levels wil be discussed later, as in most cases a fairly accurate approximation of the blood level of alcohol can be inferred from the urine result, provided it is interpreted in the light of the clinical findings.

It is possible that a little extra effort in persuasiveness might have induced a few more drivers to submit to blood tests, or that some of the more difficult and comatose drivers could have been venepunctured, but the clamour for compulsory chemical tests in various quarters loses much of its weight, so far as Brisbane is concerned, when it is shown that 91% of drivers have submitted voluntarily.

All blood samples in Brisbane are taken with a "venule" containing oxalate. The analyses are performed by the Government analyst, and all alleged drunk in charge (D.I.C.) examinees are told that they or their legal representatives may obtain the results prior to a court hearing, should they decide to plead "Not guilty". However, it is found that most drivers arrested on this charge, upon sober reflection the next day, decide to plead "Guilty". Others seek a remand pending results of their chemical tests, and the majority of these — usually on the advice of their legal representatives — decide to alter their plea to "Guilty" if the chemical result is above the widely accepted figure of 0·15% (weight for volume).

In Queensland arrested D.I.C. persons are brought before the magistrate's court and, should they plead "Not guilty", police, medical and analyst's evidence is heard and the magistrate then gives his verdict — guilty or not guilty; if the person is found guilty, the magistrate can impose a fine and/or imprisonment. Forfeiture of the driver's licence for three months at least is automatic. An appeal can be made (against the magistrate's verdict) to a higher court, but this is rare.

In England a suspected alcoholic driver, on arrival at a police station, is informed that a doctor will be called to examine him. He has the right to refuse examination and to request the police to call on his behalf a doctor of his own choice. However, in Queensland, Section 259 of the Criminal Code reads as follows:

> Examination of Person of Accused Persons in Custody.
>
> 259. When a person is in lawful custody upon a charge of committing any offence, it is lawful for a police officer to search his person, and to take from him anything found upon his person, and to use such force as is reasonably necessary for that purpose.
>
> When a person is in lawful custody upon a charge of committing any offence of such a nature and alleged to have been committed under such circumstances that there are reasonable grounds for believing that an examination of his person will afford evidence as to the commission of the offence, it is lawful for a legally qualified medical practitioner, acting at the request of a police officer, and for any person acting in good faith in his aid and under his direction, to make such an examination of the person of the person so in custody as is reasonably necessary in order to ascertain the facts which may afford such evidence, and to use such force as is reasonably necessary for that purpose.
>
> (The Criminal Code and Criminal Practice Rules of 1900—as amended as at 30th June, 1950.)

This rather suggests that here he has not this right to refuse examination if it is insisted upon, and this is supported by paragraph 1557 of the Policeman's Manual already quoted, although usually the Government medical officer called by the police will not force the issue to this extent. In such cases careful observation is usually sufficient.

If this interpretation of Section 259 is applicable, then specific legislation making blood tests compulsory is not necessary, as the powers already exist, even though it is not obligatory on the part of the police or examining doctor to use them, as in most cases it is possible to "ascertain the facts which may afford such evidence" by less forceful methods and so obviate the necessity for recourse "to such force as is necessary".

Table I summarizes the examinations and chemically tested cases for the period under consideration.

Age and Sex Distribution.

Of 2645 persons arrested, only 20 (0·7%) were females. The age distribution of the 2625 males is set out in Table II, together with the age distribution of the Brisbane male population (1954 census).

It can be seen that the age group most represented was 30-39 years. There were 921 persons (35%) in this age group, which represents only 24% of the male population of Brisbane. Again, 26% of arrested drivers were in the 40-49 years age group, who represent only 17% of the Brisbane male population.

TABLE I.

Particulars Concerning Drinking Drivers Medically Examined at City Watchhouse, Brisbane, for the Eight Years 1952–1959, Inclusive.

Year.	Numbers of Drivers Arrested (Including D.I.C. Cases).	Specimens Examined by Analyst.			
		Blood Only.	Urine Only.	Blood and Urine.	Total.
1952	158	30	6	118	154
1953	171	33	23	105	161
1954	193	27	35	126	188
1955	291	47	32	188	267
1956	353	46	51	201	298
1957	528	128	27	349	504
1958	478	87	23	322	432
1959	473	98	16	291	405
Total ..	2645	496 (18·7%)	213 (8%)	1700 (64·3%)	2409

Obviously there are relatively more arrests in the 30-39 years age group, and the probable reason is that people under 30 years of age have not yet acquired fixed drinking habits, and probably have not fully graduated to the car-owning and driving category. After 50 years of age, many have no doubt learned discretion, which suggests that any propaganda or educational campaign could well be directed to people in the early twenties, as it is at this age that the percentage of drinking-driver arrests approximates to the age-group percentage in the community. However, the peak age for motor vehicle drivers causing road accidents (excluding drivers of motor cycles) which involve property damage of more than £25 is between 20 and 29 years for all Queensland, which is some 10 years earlier than the peak age for D.I.C. arrests in Brisbane. Separate figures for Brisbane accidents were not available and unless it can be shown that the accident rate is higher outside Brisbane, the comparison is significant.

The age group most at risk is very obvious from Figure I. In this figure, drinking-driver arrests are taken in preference to drinking-driver convictions, because the vast majority of drivers had consumed more than three drinks, even though a few may have escaped conviction on a technicality.

Season and Month of the Year.

The months of the year in which 2645 arrests were made, together with the total arrests in each month, were as follows: in January, 136; in February, 182; in March, 199; in April, 236; in May, 245; in June, 223; in July, 233; in August, 227; in September, 219; in October, 244; in November, 222; in December, 279.

The distribution according to the month is not significant except that for December, when the effect of the festive season is apparent; and the drop in January and February may be due to shortage of money after the holiday expenses of Christmas. Also city traffic is lighter in January and so traffic incidents attracting police attention are fewer.

Day of the Week.

The days on which the 2645 arrests were made were as follows: on Sunday, 213 (8·0%); on Monday, 253 (9·6%); on Tuesday, 237 (9·0%); on Wednesday, 284 (10·7%); on Thursday, 308 (11·7%); on Friday, 492 (18·6%); on Saturday, 858 (32·4%).

More than half the arrests occurred on Friday and Saturday. The effect of payday and the work-free Saturday is obvious. Figure II clearly shows the trend.

Hour of the Day.

Table III shows the time of the medical examination to the nearest hour, the actual arrest having taken place on an average some 30 to 45 minutes previously. If arrests were distributed evenly throughout the day each hourly period would represent 4·2% of the total number of arrests. A column has therefore been included in Table III to show the 24-hour distribution on a percentage basis. Comparatively few examinations (only 3%) are made in the 3 a.m. to noon period, the numbers thereafter progressively increasing throughout the afternoon and reaching a peak (45%) in the 9 p.m. to midnight period. (Legal closing hour for hotels in Queensland is 10 p.m.) In the three hours after midnight 13·5% of examinations are performed. It is interesting and instructive to compare the peak period of arrests in those States that still have 6 p.m. closing of hotels. In Victoria (6 p.m. closing) the peak hour is 6 to 8 p.m. (Birrell, 1960).

TABLE II.

Age Distribution of Arrested Drinking Drivers in Brisbane and of Brisbane Male Population (1954 Census).

Age Group. (Years.)	Brisbane Male Population.		Drinking Drivers Arrested (Male).	
	Number.	%	Number.	%
15 to 19 ..	17,017	9·8	37	1·4
20 to 29 ..	33,045	18·9	592	22·6
30 to 39 ..	37,639	21·7	921	35·1
40 to 49 ..	32,585	18·6	683	26·1
50 to 59 ..	24,658	14·1	285	10·8
60 to 69 ..	18,717	10·7	68	2·6
70	10,787	6·2	14	0·5
Unstated ..	—	—	25	0·9
Total ..	174,448	100·0	2625	100·0

TABLE III.

Period of Day in which D.I.C. Cases were Examined after Arrest.

Time Examined (to Nearest Hour).	Drivers Examined.	
	Number.	Approximate Percentage of Total.
01·00 hours	97	3·88
02·00 hours	59	2·36
03·00	29	1·16
04·00	14	0·56
05·00 hours	5	0·2
06·00	4	0·16
07·00 hours	1	0·04
08·00 hours	4	0·16
09·00 hours	1	0·04
10·00 hours	5	0·20
11·00 hours	11	0·44
12·00 hours	17	0·68
13·00 hours	23	0·92
14·00 hours	26	1·04
15·00 hours	56	2·24
16·00 hours	112	4·48
17·00 hours	156	6·24
18·00 hours	187	7·48
19·00 hours	205	8·20
20·00 hours	290	11·60
21·00 hours	283	11·32
22·00 hours[1]	449	17·96
23·00 hours	408	16·32
24·00 hours	189	7·56
Unstated	14	0·56

[1] Hotels close at 10 p.m. in Queensland.

Blood and Urine Alcohol Tests.

The metabolism of alcohol and its estimation in body fluid has been well reviewed by McCallum (1954). Briefly, alcohol enters the stomach, from which up to 40% may be absorbed, and the remainder passes to the upper intestine, from which it is rapidly absorbed. Depending upon the

stomach emptying delay (usually due to the presence of food) the peak of absorption from the gut into the circulation occurs from half to one and a half hours after ingestion. Being rapidly diffusible, alcohol is soon distributed (or absorbed) in the body fluid, which averages about two-thirds of the body weight, so that when equilibrium is reached the alcohol content of the blood is a constant index of the relative alcohol content of the brain.

The alcohol in the tissues is eliminated as follows: (i) by metabolism (oxidation), mainly in the liver (approximately 90%–95%); (ii) by excretion through the kidneys (approximately 2%–5%); (iii) by excretion from the lungs (approximately 1%–2%); (iv) by small amounts in the saliva, the fæces and the sweat.

because alcohol actually stimulates diuresis. Diffusion of alcohol from adjacent tissues to the bladder and vice versa is of academic interest only and mainly concerns the autopsy pathologist.

If we know the alcohol content of the blood-stream of a person who has been arrested on a drinking-and-driving charge, then we can calculate with fair accuracy the amount of alcohol which has been absorbed from the gut, and this must be less than the amount actually imbibed. Knowing the alcohol percentage in his blood-stream — once equilibrium has been reached between the blood and the tissues (from half to one and a half hours after its ingestion) — and knowing that the alcohol is dissolved or dispersed in approximately two-thirds of his body weight

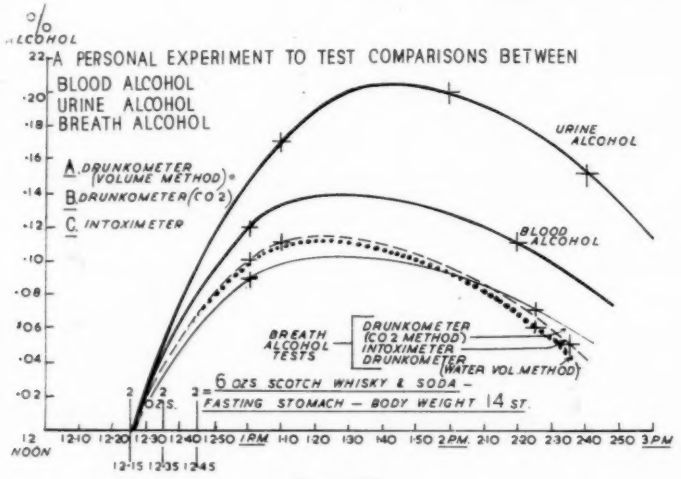

FIGURE IV.

Graph showing results of a personal experiment testing comparisons between urine alcohol level, blood alcohol level and breath alcohol.

The concentration of alcohol excreted through the kidneys is approximately that of the blood plasma, and so is a little higher than the concentration found in whole blood. In urine obtained from the bladder, the percentage represents the mean for a given period between two acts of micturition as compared with that obtained for whole blood, which represents a given moment. Alcohol is a non-threshold substance and is not selectively reabsorbed in the tubules, and so it would seem that, whilst alcohol passes in very dilute quantities through the glomeruli, its ultimate concentration in the ureter is approximately that of the blood plasma (Haggard, 1943), although with profuse diuresis it may be a little less. The relative solubility of alcohol in plasma and in urine, and the variation in concentration according to the specific gravity of urine are factors in producing a certain blood/ureteric-urine alcohol ratio at a given moment, and this ratio will differ from the blood/bladder-urine alcohol ratio when urine has accumulated for some time.

Since blood alcohol estimations are usually performed on whole blood, it follows that alcohol estimations on blood and ureteric urine collected simultaneously will give a lower figure for blood, and the ratio is found to be somewhere in the region of 1·0:1·3, though in actual practice, and particularly when the urine is a bladder sample, the ratio may vary from 1·0:0·7 to 1·0:1·7 (Ellerbrook, 1943). However, once equilibrium of alcohol in the blood-stream and tissues is reached — and the vast majority of drinking drivers have reached this stage when examined — the blood/urine ratio is fairly constant (varying from 1·0:1·1 to 1·0:1·3. A large initial reservoir of non-alcoholic urine in the bladder, diluting the renal excretion alcohol, can occur in the early stages only, and in practically all cases of actual arrest, this initial non-alcoholic reservoir has been voided prior to the arrest and medical examination, (the water content of the body), one can, by a simple calculation, derive the amount of absolute alcohol actually absorbed from the gut, and which is influencing his brain at the time of examination. This is factual information which should be, and in most cases is, readily acceptable and helpful to the courts. Simple formulæ have been devised for making this calculation. The ratio of the concentration of alcohol in the tissues (that is, actually absorbed from the gut) to the concentration of alcohol in the blood (that is, whole blood) is termed the r factor and has been calculated by Widmark as 0·68 for man. Newman and Lehman (1937) calculated it at 0·7. Newman (1941) has shown that plasma contains 30% more alcohol than the red blood cells, making the concentration of alcohol in serum about 15% higher than in whole blood. If A represents the amount in grammes of absolute alcohol in the tissues, p the body weight in kilograms, and c the concentration in the blood expressed as milligrammes per gramme (that is, the percentage of alcohol in the blood $\times$ 10), then use the formula $A = p \times c \times r$ will provide the information. For convenience, r is usually taken as two-thirds.

In many countries the blood alcohol finding is admitted as prima-facie evidence, because thousands of experiments have shown that, at certain blood levels of alcohol, the normal control of a motor vehicle and the necessary alertness in modern traffic are impaired, to a greater or lesser degree, in even the most hardened toper.

The upper limit permissible varies in different countries — from 0·08% and 0·10% in some Scandinavian countries to 0·15% in many American States.

In the United States of America, Section 54 of *The Uniform Act Regulating Traffic on Highways of the United States of America* states:

54. Persons under the influence of intoxicating liquor or drugs.

A. It is unlawful and punishable as provided in paragraph (D) of this section for any person who is under the influence of intoxicating liquor to drive or be in actual physical control of any vehicle within this State.

B. In any criminal prosecution for a violation of paragraph (A) of this section relating to driving a vehicle while under the influence of intoxicating liquor: The amount of alcohol in the defendant's blood at the time alleged as shown by chemical analysis of the defendant's blood, urine, breath or other bodily substance shall give rise to the following presumptions:

(1) If there was at that time 0·05 per cent. or less by weight of alcohol in the defendant's blood, it shall be presumed that the defendant was not under the influence of intoxicating liquor.

(2) If there was at that time in excess of 0·05 per cent. but less than 0·15 per cent. by weight of alcohol in the defendant's blood, such fact shall not give rise to any presumption that the defendant was or was not under the influence of intoxicating liquor, but such fact may be considered with other competent evidence in determining the guilt or innocence of the defendant.

(3) If there was at that time 0·15 per cent. or more by weight of alcohol in the defendant's blood, it shall be presumed that the defendant was under the influence of intoxicating liquor.

(4) The foregoing provisions of this paragraph shall not be construed as limiting the introduction of any other competent evidence bearing upon the question whether or not the defendant was under the influence of intoxicating liquor.

Some other countries recognizing blood alcohol levels are Denmark, where blood levels of 0·10% or higher result in 15 to 40 days' imprisonment; Sweden, where a level of 0·15% results in imprisonment for up to one year, and levels of 0·08%–0·15% carry a fine or imprisonment for up to six months; and Norway, where a level in excess of 0·05% is presumed to be indicative of a person's being under the influence. Opinions and experiences in these various countries are therefore fairly unanimous in accepting blood alcohol levels as indicative of impaired competence, to the extent that they have legal recognition and acceptance by the courts. The question of whether such tests should be compulsory or not is a matter of opinion. The Scandinavian countries, Switzerland, France, Czechoslovakia and some Canadian provinces have compulsion.

As mentioned earlier, there is no specific legislation in Queensland making blood tests compulsory, nor are the results admitted as prima-facie evidence. However, over the past few years most of the magistrates in Brisbane have come to admit the results of the test as evidence if such evidence is given by the examining doctor, and over 80% of arrested persons have had blood samples tested on a voluntary basis. This is similar to the position in Holland, where blood tests, although not obligatory, are practically routine. Whether or not one favours compulsion, and whether or not an arbitrary blood level, based on many thousands of experiments and experiences, is desirable,

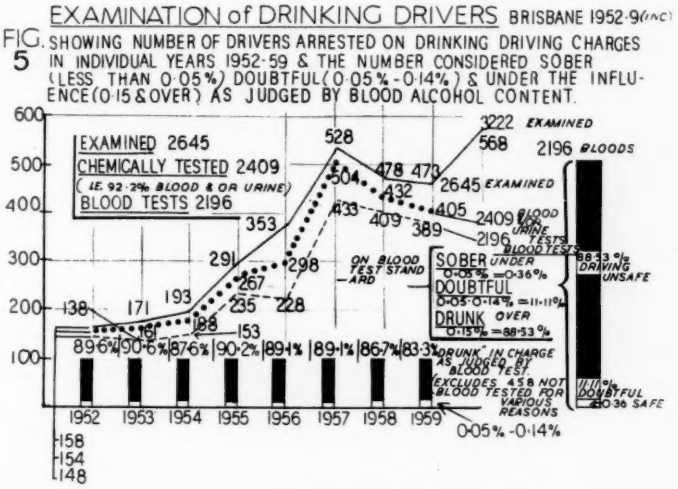

FIGURE V.

Graph showing the number of drivers arrested on drunken driving charges in Brisbane in individual years (1952-1959), including those considered sober (blood alcohol level of less than 0·05%), doubtful (blood alcohol level between 0·05% and 0·14%) and under the influence (blood alcohol level of more than 0·15%).

there can be no doubt that a knowledge of how much alcohol has been actually absorbed into the system, as distinct from the amount imbibed, must be of tremendous help to the court. This evidence should be part of the doctor's evidence and the doctor, not the court, should interpret the figures. For this, if for no other reason, blood tests should be performed whenever possible.

As there are times when it is more convenient, or more practicable, to obtain urine specimens, the question arises of the reliability of the urine alcohol level as an index of the blood alcohol level. In this connexion 1409 instances of blood and urine tests being made simultaneously are quoted here, and shed some light on the subject. As already stated, the urine was obtained without preliminary bladder emptying, as under actual working conditions this is the specimen most often procured without undue inconvenience. In only a small proportion of these cases would the accepted ratio of 1·0:1·3 have been significantly wide of the mark, and given a presumptive blood level above what was actually found on testing. However, even in these instances the urinary findings, by giving an "average" reading for the few hours prior to the taking of the blood sample, were of use in sifting apparently contradictory evidence. There can be little doubt that any person with a blood alcohol level of 0·15% can be considered to have imbibed a dangerous amount of alcohol in so far as his safe control of a motor vehicle is concerned, but a blood sample is not always available. Therefore the chemical evidence has at times to be sought from the urine. In England, blood tests are not done as a rule, as there is some apprehension concerning actions for assaults if vene-

TABLE IV.

Variation in Alcohol Content in Urine Samples at a Particular Level of Blood Alcohol in a Consecutive Series of 1409 Cases.

Blood Alcohol Level (%).	Number of Samples.	Variations in Alcohol Levels in the Corresponding Urines (%).		Averages of the Various Urines.			
		Lowest.	Highest.	Mean Average.	Median.	Mode.	Theoretical (if Ratio 1·0 : 1·3 Adopted).
0·02	1	0·02	0·02				
0·03	2	0·06	0·08				
0·04	0	—	—				
0·05	1	0·11	0·11				
0·06	2	0·09	0·10				
0·07	4	0·08	0·13				
0·08	3	0·05	0·13				
0·09	5	0·12	0·14				
0·10	11	0·12	0·19				
0·11	18	0·13	0·22				
0·12	29	0·13	0·24				
0·13	31	0·12	0·29				
0·14	39	0·16	0·29				
0·15	55	0·17	0·27				
0·16	67	0·18	0·28				
0·17	60	0·15	0·31				
0·18	83	0·15	0·32				
0·19	99	0·16	0·33				
0·20	123	0·20	0·33	0·263	0·265	0·28	0·26
0·21	108	0·22	0·34	0·285	0·285	0·28	0·273
0·22	90	0·20	0·36	0·29	0·285	0·285	0·286
0·23	119	0·22	0·41				
0·24	95	0·24	0·38				
0·25	85	0·18	0·43				
0·26	55	0·26	0·37				
0·27	72	0·26	0·41				
0·28	36	0·30	0·39				
0·29	32	0·31	0·44				
0·30	22	0·31	0·43				
0·31	27	0·35	0·44				
0·32	13	0·37	0·47				
0·33	5	0·39	0·44				
0·34	4	0·39	0·43				
0·35	1	0·45	0·45				
0·36	2	0·35	0·37				
0·37	3	0·44	0·50				
0·38	3	0·44	0·50				
0·39	—	—	—				
0·40	3	0·50	0·54				
0·41	—	—	—				
0·42	—	—	—				
0·43	1	0·59	0·59				
Total ..	1409						

puncture is performed. This objection cannot be raised to the collection of a urine sample, although in Scotland the testing of a urine sample without the patient's consent has been held to constitute theft.

The two great advantages of the urine test are (i) it represents the mean for the varying blood levels for the period between acts of micturition, provided the drinking was commenced some time prior to the first urination, and (ii) if a simultaneous blood sample is available it gives a definite indication as to whether the blood alcohol level was rising or falling at the time the sample was taken.

The disadvantages—or rather weaknesses—of the urine test are (i) the urine may be diluted with non-alcoholic urine previously present in the bladder before the drinking started, although as already stated this has usually been voided before the actual arrest and/or examination takes place, and (ii) the drinking of concentrated alcoholic beverages by a person with good bladder control could result in a significantly high alcoholic urine with an insignificant blood alcohol figure—for example, the sample voided on the morning after the night before could quite easily give a reading of 0·12% to 0·15% with a blood level of less than 0·05%. However, such instances are rare and, whilst I have found a few in experimental work, I have not as yet encountered wide variation in persons examined on drink-driving charges. (iii) The results of the test may constitute evidence against the patient unless he is informed of the test and gives consent, but this is splitting hairs, as urine is not infrequently examined for several other abnormal constituents, whose detection could be in his favour—for example, acidosis and drugs.

In the vast majority of cases the assumption that the blood level at the time of arrest, and the urine level at the time of examination were in the proportion of 1·0:1·3 or 1·4 would not have resulted in a miscarriage of justice. Table IV shows the degree of variation which I have found to occur for certain specified blood levels in my series of cases. However, from this table we see that a urine alcohol figure as high as 0·22% was seen with only 0·11% in the blood, and conversely that a person with 0·18% alcohol in his urine (and presumably less in his blood) did in actual fact have 0·25% alcohol in his blood.

The two examples—admittedly the extremes—serve to illustrate the fact that we can assume that the blood alcohol was either rising or falling at the time the specimen was taken and so a reasonable inference can be drawn as to the probable blood level one or two hours previously when the traffic incident occurred. I say probable, because one cannot be dogmatic on this point. It is a cardinal principle of British justice that a man is innocent until proven guilty, and if technicians and magistrates and judges are expected to draw rigid conclusions from a legally fixed blood level or urine level of alcohol, then justice will, not infrequently, go astray. The only person in a position, or indeed competent, to draw any fair conclusion is the doctor who conducts the actual clinical examination.

On urine results alone therefore it is not safe—at least within this range—to draw conclusions too readily, as is done in the British Medical Association Handbook.

The British Medical Association Handbook has this to say regarding the presence of alcohol in body fluids:

> Among the data which may help the medical examiner to estimate the degree of intoxication is the concentration of alcohol in the body fluids. Urine and blood are the obvious fluids of choice. Although blood is suitable, it may be difficult to collect from a person who is uncooperative and whose powers of coordination are impaired. If a mishap occurs it may lead to medico-legal complications and possible litigation. Urine serves the desired purpose equally well and has the advantage that samples are easy to collect. The Committee therefore recommends that, except in special circumstances, urine should be the body fluid used.

From the experience of these 1409 Brisbane cases in whom simultaneous blood and urine tests were collected, I consider that the statement that urine serves the desired purpose equally well is not strictly accurate, particularly as it is not recommended in the booklet that the bladder be initially emptied and a second specimen obtained after some 15 to 20 minutes. This second sample, of course, would be more reliable, but to rely on getting it in all cases is a counsel of perfection because, under conditions obtaining at the time of arrest or examination, a second specimen is often not available within a reasonable time, nor is there any guarantee that the bladder has been completely voided on the first occasion.

If blood and urine tests are to be admitted by courts as evidence, then such evidence should be accepted as significant, or prima facie, only if both blood and urine samples are collected at the same time.

Blood or urine tests alone could well be admitted, but only as relevant evidence and with greater weight being given to the result of the blood test. Thus a person with 0·15% of alcohol in his blood and with a correspondingly higher urinary alcohol level could be presumed to be in the process of sobering up at the time of his medical examination, and so could be fairly convicted without further evidence because the level was certainly not less than 0·15% at the time of his arrest, provided that the medical examination was carried out within a reasonable time—perhaps an hour—after arrest, and that there was evidence of unusual behaviour at the time of arrest.

Smith and Purves-Stewart (1932) came to the conclusion that urine analysis as a means of diagnosing drunkenness was valueless. However, this must not be taken to mean that the clinician who actually examines the patient cannot derive a considerable amount of assistance from this test, and I would again stress that it is of value chiefly for the doctor who examines the patient, and it is not for the court or some technician, chemist or pathologist to draw any conclusions from the result of a test.

Mr. Justice Little in Melbourne in a recent case advised the jury to reject the evidence of a chemical test because the person who did the test was unable adequately to relate the test and its result to the particular defendant.

However, the histograms (Figure III) show that most cases conform to a pattern.

Breath Test.

In recent years the breath test on the alleged alcoholic driver has received prominence, some hailing it as the answer to the problem, others condemning it as unreliable.

Its great advantage is that it can be taken by the police officer at the actual time of arrest—unlike the blood test, which is usually taken up to an hour later. Those who condemn it do so on two counts—firstly, that it is not a reliable index of the blood alcohol level; and secondly, that the police officer who takes it is not a trained technician, and so faulty techniques could give unreliable findings.

In Brisbane some use has been made of two types of breath test apparatus—the "Drunkometer" and the "Intoximeter"—with not very encouraging results so far. An experiment therefore was carried out under laboratory conditions and the results are shown graphically in Figure IV.

Admittedly only two points of time have been taken for blood and breath readings, but the three points of time in the urine test do give a reasonably representative curve, and unless my concept of the physiology of alcohol metabolism is wrong, the other curves should be reasonably accurate. It will be seen that the breath tests, whilst showing some consistency amongst themselves, are well below the expected figure as shown by the blood and urine curves, and also well below the expected figure calculated from the known intake.

The experiment was carried out under the best conditions. The subject was a medical practitioner, the locale was a laboratory and the actual tests were performed by experienced analysts on the spot—conditions hardly likely to be duplicated in actual practice.

A more elaborate machine employing a photoelectric cell to determine the end-point may be more accurate, but is also more costly than the "Drunkometer" and "Intoximeter" machines.

My reasonably extensive experience has convinced me that the ultimate assessment of any person's culpability is dependent on the clinical examination taken in conjunction with the result of chemical analysis, and on present experience to date I think it would be most unjust and most unscientific to admit a chemical analysis as evidence unless, at the same time, the clinician's interpretation of the analyst's results were admitted and given more importance than the chemical result.

If breath tests are to be employed in this country on any extensive scale, then they should be studied in their country of origin by an experienced clinician under actual working conditions. He should note the state of the accused and the skill of the police officer taking the test, and he should correlate these opinions with subsequent laboratory findings, including blood tests.

Breath tests are not used in England; they have been condemned in South Africa and are highly extolled in the United States of America and some Canadian States, so it would seem their final assessment must await further study and experience.

Discussion.

The more one sees of the drinking driver problem, the more one realizes the difficulty, not only of dealing with its preventive aspect, but also of making the punishment fit the crime.

A very large percentage of drinking drivers arrested are decent citizens with no thought or intent to commit a breach of the law—they simply have not realized and are ignorant of the fact that a condition of mild euphoria from alcohol has placed them within the definition of being under the influence, or that in Queensland it is not necessary even to drive or attempt to drive a car in order to "be in charge" of it. In fairness to this large body of unwitting offenders much more publicity could be given to the matter.

Again, a number of drivers who are involved in traffic incidents which are not due to any fault of their own, or in any way attributable to the small amount of alcohol they may have consumed, become panicky and agitated at the time, and despite the verdict of their doctor that they had reasonable control of their faculties when examined, nevertheless must spend at least four hours in the Watchhouse, and usually decide to plead "guilty" next day when brought before a magistrate, finding this the best and quietest way out. The practice in England is to have the police surgeon examine the driver, and if his verdict is that he has reasonable control of his faculties, his car keys are handed back to him and he goes on his way.

Also the slightly inebriated person, who realizes his condition and who parks in a secluded byway until he recovers, invariably attracts the same punishment and fine as the paranoid psychopath who hurtles his car with dangerous speed and erratic course along the crowded highway.

One wonders whether the charge might not be more fairly made were it "dangerous or inefficient driving due to alcohol", which, after all, is what the legislature is really trying to prevent. This would catch the driver unduly reckless and exuberant as the result of a couple of beers and who now goes free, and would be fair to the mature and responsible citizen who decides to sleep it off rather than to drive.

In addition, it is an offence in Queensland to drive under the influence of alcohol and/or drugs and it is extremely doubtful whether the mild exuberance in a normal person from a few drinks is not a lesser risk factor than the euphoria or casualness induced by the numerous sedatives and tranquillizers consumed daily by thousands of drivers.

The carelessness and thoughtlessness induced by neuroses and anxiety states due to marital disharmony or business stresses can make a driver reckless and impatient to a degree, when three whiskies may have restored his equanimity.

The foregoing remarks are not to be construed as condoning the actions of the drunken driver, but rather to point out that there is a tendency to attribute the mounting toll of road casualties to alcohol alone and to forget those other factors which, with poor road design, should bear their share of responsibility.

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ON DEPRESSION.

By I. PIERCE JAMES, M.A., M.B., D.P.M.,

Honorary Assistant Psychiatrist, Royal Perth Hospital, Perth.

IN recent years much has been written of the many guises in which psychiatric illness may present to the physician, and depression, with its somatic accompaniments, often plays a central theme in such writings.

Depression is somewhat of an enigma in psychiatry—for in spite of impressive advances in the therapy of affective illness there is still much controversy over, and little understanding of, its sufficient or necessary causation. A less obvious problem of depression is a semantic one. The term depression is used in three distinct senses; and yet this may not be immediately apparent from the context. "Depression" may be used to describe a patient's mood—as a symptom; it may be used to indicate a clinical illness—as a nosological entity; and yet again it may be used in the sense of a psychopathological sequence—the psychological mechanism of depression.

A large proportion of patients suffering from all sorts of illness show a predominant mood of sadness, or an affect of depression. However, to single out this aspect and to describe all such patients as suffering from depression blurs the valuable clinical distinction between affective illness (those specific psychiatric syndromes in which depression is the primary or distinctive feature) and the mood of depression which may be a symptom in many psychiatric or physical disorders—so that depression becomes more or less synonymous with emotional disturbance. In this light the present status of the concept of depression in psychiatry may be considered.

The Symptom of Depression.

One dimension of human mood is between cheerfulness and sadness. Most normal individuals react to certain life situations by becoming cheerful or sad, and this may be regarded as normal psychophysiological response. The extent (or at least the outward expression) of this mood response is to some extent socially determined. In one cultural group it may be normal to weep and express sadness in a certain situation (for example, the temporary departure of a friend or relative), whereas in another such behaviour would be most unusual. Increased lability of mood response is seen in some individuals as a permanent characteristic of their personality, or as a concomitant to the endocrine changes of adolescence, the menopause or the premenstruum. In others it may result from physical illness (for example, organic brain damage or chronic drug intoxication) or psychiatric illness. Lability of mood may thus have a cultural, a temperamental or a pathological basis. This lability of mood response to the immediate environmental situation is to be distinguished from the prolonged cyclic mood changes of the constitutional cyclothymic personality.

If the mood change is even more marked we talk of depression. Then other qualities of feeling-tone in addition to sadness become manifest. Not only is the patient sad, but his thought and behaviour are dominated by his mood. Gutheil (1959) describes depression as consisting of sadness plus pessimism. In more profound states of depression (such as are seen in the manic-depressive psychoses) psychomotor disturbance—retardation or agitation—may dominate the picture.

Depression of affect may not be assumed because the patient looks tired, dejected or preoccupied or answers in the affirmative to the question: "Do you feel depressed?" He should be asked: "What do you mean by depression? Can you describe it as fully as possible?" Depression means different things to different people. Some individuals will describe tension, confusion, lassitude, preoccupation with obsessional thoughts or even headache as depression. The physician must make certain that the patient is describing depression of mood.

A depressed mood is a common symptom of physical or psychiatric illness. It may be predominant in toxic states, as with infections (typhoid fever or influenza), after the taking of drugs (sulphonamides, reserpine, barbiturates or alcohol) or in jaundice and uræmia. It may accompany metabolic disorder (myxœdema, Addison's disease or premenstrual tension) or such diseases as brain tumour, pernicious anæmia and diabetes. In psychiatric illness some degree of depression is the rule rather than the exception. It occurs not only as the basic psychological state around which accessory symptomatology may evolve in the affective reaction syndromes (manic-depressive states and reactive or "neurotigenic" depression), but even more frequently as a symptom in many types of neurosis, psychosis or personality disorder in which the basic mood change is of less psychopathological importance.

There are several dimensions of mood disturbance. The normal person fluctuates between normal sadness and cheerfulness. Exaggerated responses are depression and elation, characterized by sadness plus pessimism on the one hand, and by cheerfulness plus optimism on the other. The psychotic patient experiences melancholic depression and hypomanic elation, characterized by depression plus psychomotor disturbance and elation plus psychomotor disturbance respectively.

Depressive Illness.

In this sense the term depression or depressive illness is applied to those psychiatric syndromes in which depression of affect is the primary or central feature. It is customary to distinguish between endogenous and reactive depressive illness on the one hand, and between psychotic and neurotic depression on the other. Endogenous or autonomous depression (as typified by manic-depressive depression or involutional melancholia) is held to arise in genetically or constitutionally predisposed individuals as a primary disturbance of mood unrelated to the immediate external environment. Reactive depression, conversely, arises in neurotically or otherwise predisposed individuals as a direct sequel to certain forms of life stress. Endogenous depression is usually severe, accompanied by psychomotor agitation or retardation and, although the mood change is primary, the evolution of secondary symptoms — delusions of guilt, hypochondriasis, suicidal

impulses, and so on — is the rule (that is, the illness is usually of psychotic intensity, or psychotic depression). Reactive depression is usually milder and unaccompanied by psychomotor disturbance or such secondary symptoms (that is, it is of non-psychotic intensity, or neurotic depression).

Although this classification is adhered to in most contemporary textbooks of psychiatry, many authors (Lewis, 1934) have pointed out that an absolute distinction between endogenous and reactive depression is neither clinically nor ætiologically valid. There is certainly no doubt that a large proportion of patients with depressive illness show a varied admixture of endogenous and reactive features, or that mainly endogenous depressions may be mild and mainly reactive depressions severe (psychotic). However, the distinction persists, as it does have a certain descriptive value and therapeutic implication.

Another point of confusion arises from the use of the term neurotic depression. This is sometimes used to indicate the severity of the disturbance (neurotic depression as opposed to psychotic depression), and sometimes it is used synonymously with reactive depression to imply that the condition has arisen as a neurotic response to certain forms of stress in particular individuals. It might be better if the term non-psychotic depression was used in the former sense and neurotigenic depression in the latter (see Figure I).

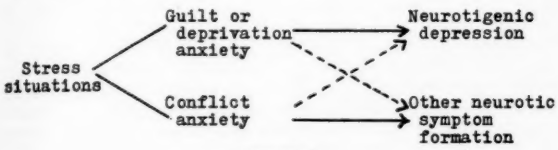

FIGURE I.

Depression of mood is a common symptom in all sorts of psychiatric illness, and it should be remembered that in only a small proportion of psychiatric patients is the diagnosis of depressive illness justified.

Roth (1959), for example, has recently described the "phobic-anxiety depersonalization syndrome", in which phobic-anxiety, depersonalization and varying degress of depression of affect occur with or without obsessional, hypochondriacal, hysterical and other symptoms. He talks of it as a pan-neurosis occurring in individuals of rigid obsessive dependent personality in circumstances of threat to their security. He regards the condition as a distinct entity, to be differentiated from depressive illness, and found that the majority of patients with the syndrome were made worse by electro-convulsive therapy.

The Mechanism of Depression.

In this sense depression is viewed as a response or adaptation to constitutional or environmental factors. The multiplicity of hypotheses offered indicates our essential lack of knowledge of the causation of depression.

Whilst it is generally accepted that there may be an hereditary predisposition to develop manic-depressive psychosis and the cyclothymic temperament (probably as the result of a "single autosomal dominant of weak and variable expression"—Meyer-Gross *et alii*, 1954), this cannot be said to account for depressive illness.

Theories are generally of three types—those which aim to account for the emergence of depression as a sequence of events (and are usually applied to the circumstances of the precipitation of neurotigenic depression), those which view depression as a biological or learned adaptive mechanism, and finally those which are concerned with the psychological predisposition of an individual to develop depression (and are generally applied to the symptom-content of "endogenous" depressive illness).

Most theories of neurosis are based upon the concept of conflict or frustration anxiety. Certain stressful situations are held to evoke neurotigenic anxiety — and neurotic symptom formation is the result of various mental mechanisms (conversion, dissociation, obsessive symptom formation, and so on, at one level; denial, displacement, reaction formation, and so on, at another) which are more or less successful in dealing with this anxiety threat to psychological homœostasis. Thus varying admixtures of acute anxiety and neurotic symptoms result as a maladaptation to the life stress. The form of the neurosis will depend upon the nature of the stress and upon the constitutional make-up of the patient (in the sense of genetic predisposition as moulded by developmental and environmental factors).

In reactive (neurotigenic) depression, depression of mood with or without overt anxiety is the main feature of the response. Most patients who come under medical care with this syndrome will have shown a tendency to develop depressive or other neurotic symptoms previously in the course of their life history. Indeed the absence of such a history or of a constitutional predisposition to depression in an apparent case of severe reactive depression will tend to cast doubts on the diagnosis and suggest the possibility that some other psychopathology (for example, schizophrenia) or pathology (for example, early dementia) may be present. Various hypotheses have been put forward as to why depression should be the response of a particular individual to a particular form of stress. Gutheil (1959) writes: "These patients have been sensitized to traumatic events by losses sustained in the past; by insecurities and unendurable situations of stress to which they were exposed in childhood." Because of previous insecurities, some individuals over-evaluate the importance of (elevate to the status of a love-object) social position, financial security and so on. Any real or symbolic threat of deprivation of this object leads to deprivation-anxiety and, as a result, to depression rather than any other neurotic reaction. Noyes (1948) talks of "guilt-producing anxiety" as occurring in situations which evoke unconscious guilt over hostility felt towards love-objects and so on. In the setting of guilt a "turning-in" of hostility leads to depression. The form of the neurosis will depend upon both the stress-producing situation and individual personality determinants (see Figure II).

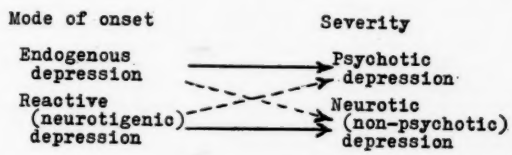

FIGURE II.

Some writers see the severely depressed state with its withdrawal and physiological hypofunction as an adaptive (or maladaptive) response to biological stress. Thus Lewis (1934) considered depressive states as a paradigm of adaptive reaction of the organism to an intolerable situation.

> Meyer suggests that depressive reactions, by withdrawing the individual from an ill-adjusted situation, serve a protective device. With this withdrawal occur the accompanying manifestations of inhibitions at various levels—vegetative, motor, affective and that of ideational content and its expression (Noyes, 1948).

Others, taking learning theory as a starting point, maintain that neurotic behaviour can become a learned response to the stimulus situation which evoked it. If the depressive reaction succeeds in reducing the drive strength in some sphere (that is, by reducing tension, solving social situations, and so on), then depressive behaviour may persist as learned non-adaptive behaviour. Eysenck (1955) includes depression under the dysthymic (introverted neurotic) states as opposed to the extraverted neuroses (hysteria, psychopathy, and so on). Thus, introversion (related by Eysenck to a constitutional or acquired predominance of central excitation over inhibition) as a

personality characteristic would predispose to the development of neurotigenic depression rather than to other forms of neurosis.

Finally there are those views which are concerned with psychological predisposition to the development of depressive symptoms—feelings of unworthiness, delusions of guilt, suicidal impulses and so on. Cameron (1947) writes that the patient

> . . . is one who has been rendered in childhood reaction sensitive to signs that he is considered inadequate, inferior, unworthy and guilty. As a child he learned that these signs heralded frightening threats to his personal security and frequently ended in rejection, condemnation and punishment by his elders. It is this sequence of selective reaction-sensitivity, threat, anxiety and hostility towards his own behaviour and himself that he carries over into his adult reactions and self-reactions . . . He is overtrained in taking the part of the hypercritical, unforgiving parent towards his own behaviour.

The psychoanalytic (Freudian) viewpoint is somewhat extensive as it has been restated in various ways by different writers. Three factors are involved: pregenital fixation, narcissistic orientation and actual or symbolic loss of an object. Infantile trauma may lead to childhood depression, pregenital fixation to over-concern with oral needs, food, money, obstinancy, hypochondriacal self-righteousness, and so on, and introjection of ambivalent love-feelings to egotism and self-destructive tendencies. Actual or symbolic loss of a love-object in later life may reactivate the depressive potential. The many extensions of these themes cannot be summarized briefly and the interested reader is referred to the psychoanalytic literature.

More recently a number of psychiatrists of the European phenomenological schools have been influenced by existential philosophy after the writings of Binswanger, Jaspers and others. This movement is particularly concerned with man's "existence". Man is held to be unique in the biological world by his ability to grasp the consequence of the realization that one day he will cease to exist. With this realization he can become aware of his "existence" and consequently his experience of being-in-the-world or *dasein*. Existence is used in the sense of *ex sistere*, to emerge. Man is seen in terms of his self-conscious awareness of his existence (his state-of-being-in-the-world) and of his need (*Verantwortlichkeit* or self-responsibility) to emerge from his state-of-being (*sein*) to his state-as-it-ought-to-be (*sein sollen*). In this are involved the many ethical and philosophical problems of "existence". Frankl (1948), writing on melancholia, maintains that any psychogenic factors can only be precipitating and that the basis is a metabolic disturbance—a physiological *baisse*. Out of the physiological *baisse* it is man who makes his melancholic experience. The physiological *baisse* leads to an insufficiency-feeling (*Insuffizienzgefühl*) which is experienced in relation to the task of emerging from *sein* to *sein sollen*. He experiences the gulf between his person as-is and his ideal as-ought-to-be out of all proportion as a conscience-anxiety (*Gewissenangst*). The conscience-anxiety makes him value-blind to himself and to his environment, but above all evokes the realization that even if interminable existence were possible, the guilt could never be atoned for. No longer has the world ethical meaning, and all must be destroyed. This catastrophe-anxiety (*Catastrophangst*) is the underlying mode of existence in melancholia.

Whilst it may be felt that the point of departure of the existential movement from orthodox psychiatry is based upon a philosophical *non sequitur* and that the coining of new phrases does not automatically provide psychological insight, there is no doubt that the phenomenological approach has added much to our understanding of the psychotic world as the melancholic sees it.

The Classification of Depression.

In its present state psychological medicine seems best served by an eclectic approach. Granted the validity of many of the objections to any classification which distinguishes between two types of depressive illness—endogenous and reactive—some such division does seem to have clinical value, provided that it is remembered that the emphasis is upon the relative importance of the variables in a given case and not upon absolute nosological distinction.

It seems useful to distinguish between primary dysphoria, secondary dysphoria and symptomatic dysphoria.

Primary dysphoria includes those cases in which the depressive mood change, usually with psychomotor disturbance, appears to be the primary process. Other symptoms (feelings of guilt or unworthiness, hypochondriacal delusions and so on) seem to follow as secondary manifestations. The depression is often, but by no means always, severe (psychotic); hereditary or personality predisposition is usually evident and external precipitating events may or may not be obvious.

Secondary dysphoria includes those cases in which the depression, although dominant, is more obviously part of a personality response to biosocial stress. The depressive mood change is usually less severe (non-psychotic). There is often a personality predisposition to neurotic response or lability of mood, but rarely a history of cyclothymia or familial affective illness. Other symptoms (irritability, fatigue and hypochondriacal concern) may be regarded more as part of the response to stress than as arising directly from a primary mood change. Secondary dysphoria may be physiological (as in grief-reaction), but it is more often encountered as an emotional response to particular forms of neurotigenic stress which have a symbolic deprivation threat for the individual.

Symptomatic dysphoria is a very much more frequent form of clinical depression in all types of practice. Some depression of mood is common in anxiety neurosis, obsessional states and many other psychiatric syndromes. Any tendency to ignore the basic psychopathology and to make a diagnosis of depressive illness simply on the presence of symptomatic depression should be resisted. Electroconvulsive therapy is a successful form of treatment of primary dysphoria (endogenous depression), but this is true only if the diagnosis is critically made, and it is generally contraindicated in neurotic states. If electroconvulsive therapy is regarded as the first therapeutic choice in psychiatry—then a diagnosis of depressive illness may be a convenient therapeutic rationalization. However, if the diagnosis is not justified the therapeutic outcome is not improved.

The relationship of depression to schizophrenia is more complex. The fundamental symptoms of schizophrenia (Bleuler, 1911) include disturbances of thought, volition and affect. Affect in this context applies to the quality of emotional response to the environment which may be altered in character or intensity—or in relation to other psychic experiences. Blunting or incongruity of emotional response is to be distinguished from disturbance, restriction or lability of the depression-elation axis. On the other hand depression of mood may be a secondary symptom development in schizophrenia. Indeed, quite severe depression is not infrequently an early symptom in hebephrenia, but it is essentially a secondary manifestation to the insidiously developing, and usually less obvious, schizophrenic disturbance. The term schizo-affective psychosis is often applied to those cases which show features of both schizophrenia and manic-depressive psychosis. The more usual mood disturbance is of mania or hypomania, but essentially depressive cases also occur. Those illnesses with an acute onset and apparent precipitation carry a good prognosis, but others persist, running truer to either schizophrenia or to manic-depressive illness in the course of their natural history—although Mayer-Gross *et alii* (1954) write: "Actual combinations of manic-depressive and schizophrenic disorders can be traced to a combination, in the patient, of schizophrenic and affective genetic factors coming from different sides of the family."

Disturbances of mood may thus be summarized. In primary dysphoria there is a primary mood change, and the depression is usually severe, with psychomotor disturbance. Secondary symptoms follow as a result of the mood change. Precipitation may or may not be apparent. A genetic, constitutional and temperamental predisposition is the rule. This occurs in endogenous depression, manic-depressive psychosis, depression and involutional melancholia, and so on.

Secondary dysphoria may be physiological or neurotigenic. The physiological type is a normal response to a life situation. Temperamental and cultural factors are involved. This includes grief reactions and is sometimes called adaptive reactive depression. In the neurotigenic type, life stress evokes guilt or deprivation anxiety and depression. Accessory symptoms are more related to the stress situation. A constitutional and personality predisposition exists. This is a neurotigenic depression, or neurotic reactive depression.

Symptomatic dysphoria occurs in medical illnesses, such as those due to infective, toxic, metabolic and other causes. It also occurs in psychiatric syndromes, being common in neurotic, psychotic or personality disorders.

Schizophrenic dysphoria occurs as a primary disturbance of affect, causing blunting or incongruity of emotional response, as a secondary symptom and as a "schizo-affective" psychosis.

The clinical distinction between endogenous and reactive depression upon the quality of the mood change or upon the content of the symptomatology has been a controversial issue. It has been said that endogenous cases may be distinguished by the occurrence of symptoms suggestive of physiological malfunction, by the presence of delusions of guilt or by the recognition of vital depression, by which term "attention is drawn to the deeper stratum of the personality involved in the endogenous depression, a stratum closely connected with the somatic concomitants of the affect" (Mayer-Gross *et alii*, 1954). However, these qualities distinguish more between "acute severe depression and chronic mild depression" (Lewis, 1938) than between differences of psychopathological causation. Certainly some primary depressive episodes in the manic-depressive patient are mild—although Arieti (1959) maintains that even here a psychotic quality can be observed which does not occur in neurotigenic depression: "The manic-depressive patient does not fight his disorder, as does the neurotic, but lives in it, and therefore is psychotic."

Even so, there do seem to be some features which may be of clinical value in assessing depressive illness, although they cannot be regarded as "pathognomonic" of one or other type of depression.

In primary (endogenous) dysphoria the disturbance of sleep rhythm is often characterized by early morning waking. The patient may wake suddenly in the early hours of the morning and is unable to sleep again. He is almost at once aware of his depression, which remains constantly present throughout the day or shows a tendency to evening remission and is but little influenced by social circumstance. If psychomotor agitation is present, this, too, tends to be constant without diurnal variation, or to decrease in the evening.

In secondary (neurotigenic) dysphoria, on the other hand, the insomnia is usually characterized by a difficulty in getting off and by fitful sleep. The patient may lie awake for hours preoccupied with memories and anxieties. By day depression and anxiety tend to become progressively worse as the day wears on, but are related to environmental events. This latter pattern is, indeed, common in the psychoneuroses generally and may be regarded as indicative of the neurotic response, whereas the former pattern is most unusual outside primary depressive illness. However, clinical assessment must always take into account not only the symptomatology, but also the constitutional background, and above all the patient's characteristic pattern of adjustment to life.

In primary dysphoria, early morning waking is the rule, whereas in secondary dysphoria the patient has difficulty in getting to sleep. In primary dysphoria, depression and psychomotor disturbance are constant, or tend to evening remission, but have little relation to the environment. In secondary dysphoria the depression and anxiety grow worse as the day progresses, but are related to social environment. Predisposing factors to primary dysphoria include a history or family history of cyclothymia, manic-depressive illness or suicide, and perhaps a rigid, authoritarian, obsessional personality. In secondary dysphoria the predisposing factors include lability of mood and an inadequate, dependent or neurotic personality; constitutional factors may be present. In primary dysphoria a good response is shown to electroconvulsive therapy and to "Imipramine". In secondary dysphoria a poor response is obtained to electroconvulsive therapy (and "Imipramine"), but monoamine oxidase inhibitors are claimed to be effective.

The Diagnosis of Depressive Illness and the Medical Problem of Suicide.

The importance of the early recognition of depressive illness rests on the efficacy of the therapeutic agents (electroconvulsive therapy, thymoleptic drugs, and so on) now available to alleviate the patient's often intense distress and also upon the ever-present danger of suicide. This particularly applies to the primary endogenous cases—although suicide is by no means confined to endogenous depression.

The patient with a neurotigenic depression usually recognizes his distress and unhappiness as of psychic origin and seeks medical aid on this account.

However, the patient with an endogenous depression, by the very nature and mode of onset of the primary mood disturbance, and with the emergence of delusions of guilt and self-blame, is often unable to accept the psychiatric nature of his condition. He lives in it, feeling that he has brought calamity upon himself by his own unworthiness. He does not complain of depression or psychological distress, because he does not recognize it as such. In this setting it is often with complaints centred upon the somatic accompaniments of depression that the patient visits his doctor. These symptoms stem from the physiological hypofunction and psychomotor disturbance which usually accompany and often dominate primary depression. Insomnia, anorexia and weight loss are common early symptoms. There may be headache, muscle pains, paræsthesia and generalized fatigue. Gastro-intestinal malfunction may lead to dyspepsia, abdominal discomfort or constipation. Menstrual disorder, impotence or diminished sexual interest are frequent. In agitated cases sweating, palpitations and precordial distress may form the presenting complaints.

Not infrequently the somatic symptoms form the basis of hypochondriacal delusions so that the patient believes that certain functions are totally lost. He may believe that he is quite unable to sleep, to pass urine or to open his bowels, although in fact these functions are only partially affected. Indeed so convinced—and convincing—may the patient be that cystoscopy, sigmoidoscopy, or even laparotomy is occasionally performed before the nature of the condition is disclosed.

The recognition and assessment of the severity of the underlying depression may present a certain difficulty. As Mayer-Gross *et alii* (1954) wrote:

> The depth of the affect cannot easily be measured from its outward expression. The silent shedding of tears may be seen in an otherwise expressionless face; another patient will mock at himself and at his complaints with a grin and sardonic but surprising humour or call himself a fraud and a fool: in another a sudden smile or expression of gaiety will deceive the physician about the severity of the underlying emotion.

However, sympathetic and careful inquiry into the patient's feelings, thoughts and attitudes will usually disclose the depressive mode of psychological functioning

beneath any social façade. Not only is there a depression of mood, but also a general reduction of mental functioning. Attention is withdrawn from the external world and the patient becomes preoccupied, inattentive and uninterested in life. He may find it difficult to concentrate, to plan his day or to cope with his work. Tasks previously carried out with enthusiasm and ease become boring and almost impossible. There is a general reduction of emotional response to events. He can become neither happy nor sad and may complain that he has lost all capacity for feeling. As the depression becomes more severe the element of pessimism pervades his life. All experiences become painful and every incident is seen as foreboding further calamity. Feelings of unworthiness, self-blame and guilt intrude and he sees his state as an inevitable and justified outcome of his previous foolishness or wickedness. Incidents from the distant past (the failure to repay a trivial debt or a premarital indiscretion) may become the centre of constant rumination and self-reproach. Everything is related to his melancholic "conscience-anxiety". Finally, projection may lead to the development of paranoid delusions (for example, that he is being sought by the police for his imagined crimes) or to hallucinations (he may notice a foul smell emanating from his body or hear the accusing voices of his neighbours), but this step is rare.

Often he feels that his guilty secrets must be hidden from his family, his colleagues and even from his doctor. In this world of isolation and hopelessness suicide may be the only choice. At this stage advice to "pull yourself together" or to "forget about your worries" may be frankly dangerous. To a patient already convinced of his utter unworthiness such advice may merely fan his guilt, increase his feeling of isolation from human society and precipitate suicide.

It is well recognized that occasionally a depressed patient who is seemingly responding to treatment attempts suicide as he improves. At the height of a depression psychomotor retardation may be such that the patient is too inhibited to carry out the act—but this retardation may decrease before the suicidal thoughts recede. Hence the need for careful supervision during therapy.

Suicidal depression is more common in the endogenous cases, and any features of this type of depression (a history or family history of cyclothymia or primary depression, or a pattern of early morning waking or evening remission, and so on) will warn of the possibility of such severity, but no sharp rules can be laid down and every case must be assessed on its own merits.

There is little doubt that depressive illness carries a far greater risk of suicide than any other form of physical or psychiatric illness, but it accounts for only a proportion (perhaps between one-third and one-half) of the suicidal acts or attempts which occur. The problem of the prediction of suicidal behaviour should not be underestimated, nor simply equated with the problem of the recognition of depression. The a-posteriori assumption that anyone who attempts self-destruction must have been depressed—and that this should have been recognized—is untenable. Stengel (1960) writes:

> Human behaviour usually has multiple motivations, not all of them obvious and some antagonistic to each other. People according to some psychiatrists, either want to die or to live. That most people who commit suicidal acts want to do both at the same time, and that these suicidal acts may serve as punishment for others seems difficult to grasp. Yet there is ample evidence that this commonly happens.

Capstick (1960) found that only 39% of 881 individuals who committed suicide had appeared depressed before the act and only 9% had voiced suicidal ideas. Harrington and Cross (1959), examining 102 cases of attempted suicide, regarded 25% as suffering from endogenous-type depressions, 27% from reactive depression (although some patients so classified "showed little or no psychiatric abnormality") and the remainder from "psychopathic personality" (11%), hysteria (9%), schizophrenia (8%), chronic alcoholism (6%) and "situational disturbance" (13%). Sixty-one *per centum* of these attempts were "impulsive and unpremeditated"; 9% of the patients had contemplated suicide for under one week and 16% for longer.

The writer's recent experience accords with this. Of 15 patients who had attempted suicide admitted to the wards of a general hospital, only three could be regarded as suffering from severe primary depression. Of the remainder, less than half showed any degree of secondary or symptomatic depression and many attempts were impulsive acts (often carried out under the influence of alcohol) by individuals of unstable "psychopathic" personality. The severity of the attempt was not directly related to the psychiatric diagnosis. Two of the patients had been attending psychiatric out-patient clinics and had been seen there within one week before the incident. Reexamination of the psychiatric data did not provide any special clue which might have anticipated the event (except in so far as such behaviour is not altogether unexpected in a large proportion of patients attending psychiatric clinics).

However, there may well be essential differences between groups of patients who commit suicide and groups of those who attempt it unsuccessfully. Only 5% of a successful group (Capstick, 1960) had attempted suicide before—whilst under 5% of those who make an attempt subsequently commit suicide (Stengel, 1960). Stengel writes of "two different but overlapping populations". One may suspect that patients with severe depression form a higher proportion of the successful than the unsuccessful group.

The annual suicide rate in most western countries is in the region of 10 per 100,000 of the population, and suicidal attempts are perhaps six or seven times more frequent (Stengel, 1960). Shepherd *et alii* (1959) found that 70% of a population sample visited their doctor at least once during the year and that 14% of these did so with overt psychiatric disorder or emotional dsturbance. In the hundred thousand population unit many of the 10,000 patients who visited their practitioner with psychological abnormality during the course of the year will show symptomatic depression and a number more severe depressive illness. Undoubtedly it is this population at risk from which the majority, but not all, the 10 annual successful and 70 unsuccessful suicides will be drawn. Lewis (1938) points out that "one cannot send into a mental hospital every doubtful case of suicidal risk", and, unless a sizeable percentage of this group are to be regarded as constant suicidal risks, it has to be admitted that in our present state of knowledge a fair proportion of suicidal acts are unpredictable.

Whilst it behoves every physician to be constantly aware of the problems of affective illness, of the many cryptic ways in which depression may present and of the importance of its early recognition and management, we can only exercise our clinical acumen to the best of our ability. Medicine is not best served by the instillation of such medico-legal anxiety into the mind of the physician that every emotionally disturbed patient is automatically to be regarded as a serious suicidal risk. It is far better served by the realization that careful mental examination and understanding inquiry of the patient's distress as he sees it will enable the correct decision to be taken in almost every case. Beyond that is beyond the limits of the art or science of medicine.

Summary.

Confusion may arise because the term depression is commonly used in three senses—to describe a symptom, an illness and a psychopathological mechanism. The current status of the concept of depression is briefly discussed in this light.

Some degree of depression of mood is common in many types of illness. However, this should be distinguished from depressive illness—those psychiatric syndromes in which depression constitutes the primary or central feature. It is clinically useful to differentiate between

primary (endogenous) and secondary (physiological or neurotigenic) depression in these syndromes, although the distinction is not absolute.

Because of its very nature, depressive illness may present to the physician in the guise of somatic illness, and it is of considerable importance to be constantly aware of this possibility. Depression carries a greater risk of suicide than any other medical condition, although suicide is by no means confined to depressive illness.

Acknowledgements.

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A PILOT CLINICAL TRIAL OF "PERSANTIN".

By IAN G. LYALL, M.R.A.C.P., AND A. J. BARNETT, M.D., M.R.C.P., F.R.A.C.P.,

From the Baker Medical Research Institute, Alfred Hospital, Melbourne.

THE relief of angina pectoris is an ever-present problem. Glyceryl trinitrate may relieve minor attacks, but it is often ineffective if these become severe, and the prevention of angina by the long-acting nitrates is very disappointing. Therefore, any new remedy which is claimed to relieve this distressing symptom requires serious consideration. Such a claim has been recently made for a new drug known as "Persantin" or "R.A.8" (2,6-bis(di-ethanol)-4,8-dipiperidino-pyrimido (5,4-d) pyrimidine).

Kadatz (1959), in experiments with anæsthetized dogs, noted that the coronary blood flow increased by 115% after an intracoronary injection of this drug. Bretschneider *et alii* (1959), in similar experiments on dogs, noted a mean increase in coronary sinus outflow of 200% during the ten-minute period of the drug's maximum activity. Fischer and Fiegal (1959) observed a definite improvement in 80% of their patients within 36 hours of the commencement of treatment with an oral dose of one or two tablets (12·5 to 25 mg.) taken three times a day, with the occasional substitution of intravenous injections of "Persantin" during the first week. Hamm *et alii* (1959) reported symptomatic improvement in more than two-thirds of 57 patients suffering from angina pectoris treated with the drug.

However, Foulds and McKenna (1960) found that "Persantin" did not produce better results than a control tablet. We therefore thought it of interest to report the results of a pilot study carried out by us prior to the latter report.

"Persantin" Given Intravenously.

Six patients with classical angina pectoris were selected. An electrocardiogram was obtained in each case; then the patient walked repetitively over two steps each eight inches high at his own pace until he had either made 25 circuits of the steps, or until angina occurred; a second electrocardiogram was then recorded. The patient then rested for 30 minutes, after which interval he received 10 mg. of "Persantin" by intravenous injection, and after a five-minute interval the above procedure was repeated.

The results are shown in Table I. It is apparent that an injection of "Persantin" neither prevented angina pectoris nor altered its duration; also, it did not cause any significant favourable changes in the electrocardiograms.

"Persantin" Given Orally.

Twelve patients suffering from angina pectoris were invited to participate in this trial. They each received two placebo tablets three times a day for two weeks and then two tablets of "Persantin" three times a day for three to four weeks. Each patient was asked to keep daily records of the number of attacks of angina and of the number of tablets of "Trinitrin" required to control them.

The results are summarized in Table II. This shows the average number of anginal attacks per day during the entire periods of treatment with "Persantin" and with the placebo. Similar figures for the second week of each treatment are also given (in order to show up a possible cumulative effect of "Persantin" not apparent until the second week). It is apparent that there was no significant clinical benefit in either case.

Discussion.

In our series "Persantin" did not prevent angina either when given orally or intravenously.

These results are at such variance with those of certain other workers (Fischer and Fiegel, 1959; Hamm *et alii*, 1959) that we sought for some explanation.

Our subjects were broadly representative of patients suffering from angina pectoris; they all had electrocardiographic changes of coronary artery disease and did not seem to differ significantly from those studied by other workers.

The dose used was the same as that recommended by the other workers. In certain of the reported cases in which no response was obtained to oral therapy, an effect was claimed for intermittent intravenous therapy given for a few days and followed by oral therapy—a method not used by us. However, Kadatz (1959) and Beisenherz (1960) have shown that the drug is rapidly absorbed after oral administration; the latter worker found that when given intravenously it is rapidly excreted and the serum concentration falls to a low level in one hour. We therefore find it difficult to believe that the failure to obtain an effect in our cases was due to failure of absorption of the drug.

Hamm *et alii* did not use a placebo, which they considered ethically unjustified, and some allowance must be made for placebo effect in their cases. We find it difficult to explain the difference between their result and ours purely on this basis, as we found angina still occurred during placebo treatment.

In conclusion, we have been unable to substantiate the claims by other workers regarding the value of "Persantin" in angina pectoris and can find no explanation for this in our method. Our results indicated that there was no case for an extensive clinical trial of this drug.

TABLE I.

Effect of Intravenous Injection of "Persantin" on Step Test Performance.

Case.	Number of Steps Taken.		Duration of Post-Exertion Angina.		Favourable Changes Produced by "Persantin" on a Post-Exercise Electrocardiogram.
	Without "Persantin".	With "Persantin".	Without "Persantin".	With "Persantin".	
I	25 (angina).	25 (no angina).	30 seconds.	—	None.
II	16 (angina)[1]	18 (angina).	2 minutes 30 seconds.	2 minutes 30 seconds.	None.
III	21 (angina).	19 (angina).	4 minutes 25 seconds.	2 minutes 50 seconds.	None.
IV	20 (no angina).[1]	25 (angina).	—	2 minutes 40 seconds.	None.
V	21 (angina).	25 (angina).	9 minutes 5 seconds.	7 minutes 10 seconds.	None.
VI	25 (no angina).	25 (no angina).	—	—	None.

[1] Step test stopped because of headache.

Summary.

A pilot clinical trial of the recently released coronary vasodilator drug "Persantin" is outlined.

"Persantin", administered intravenously in a 10 mg. dose five minutes before a modified step test, did not prevent angina pectoris in patients with proven coronary artery disease.

"Persantin", administered orally in a dose of 25 mg. three times a day for a period of three to four weeks, did not alter the incidence of angina pectoris as compared with a placebo control in a series of 12 patients.

TABLE II.

Average Daily Number of Anginal Attacks.

Case.	Average Daily Number of Anginal Attacks. During Complete Period of Trial.		In Second Week of Trial.		Conclusion.
	With Placebo.	With "Persantin".	With Placebo.	With "Persantin".	
VII	9	8	11	8	No significant clinical benefit.
VIII	1	2	1	2	No significant clinical benefit.
IX	5	6	6	5	No significant clinical benefit.
X	1	1	1	0	No significant clinical benefit.
XI	2	3	0	0	No significant clinical benefit.
XII	2	2	2	2	No significant clinical benefit.
XIII	0–1	0–1	0–1	0–1	No significant clinical benefit.
XIV	3	5	4	3	No significant clinical benefit.
XV	1	2	1	1	No significant clinical benefit.
XVI	3	5	3	4	No significant clinical benefit.
XVII	1	1	1	1	No significant clinical benefit.
XVIII	4	4	4	4	No significant clinical benefit.

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Reports of Cases.

A DRAMATIC SIDE EFFECT OF A NEW DRUG, "LIBRIUM".

By ALLEN A. BARTHOLOMEW, M.B., B.S. (Lond.), D.P.M. (Lond.),

Psychiatrist Superintendent, Alexandra Clinic, Melbourne, Victoria.

A NEW SUBSTANCE, "Librium" (7-chloro-2-methylamino-5-phenyl-3H-1,4 benzodiazapine-4-oxide hydrochloride), has become one of the popular drugs now in use. Not only is it being used for the reduction of anxiety in a number of conditions, but it is considered by some to be the drug of choice, in fact almost specific, in the treatment of alcoholism (Speight, 1960; Lawrence, 1960; Lawrence *et alii*, 1960).

Drowsiness as a side effect of the administration of "Librium" was noted by Lawrence (1960), Lawrence *et alii* (1960) and Kinross-Wright *et alii* (1960), who observed that it was not uncommonly seen after 4 to 10 days of treatment, that it occasionally occurred with ataxia and that dizziness was also to be noted; Farb (1960) commented upon it in 3 of 45 patients; Breitner (1960) wrote of "occasional drowsiness" and "dizziness and some cases reported falling and hurting themselves", and Thomas (1960) records "sluggishness" in about 10% of his cases. Robinson (1960) noted "excessive drowsiness was observed in 8 patients" out of a total of 185, and Ticktin and Schultz (1960) noted "sleep often occurred following administration of the drug . . . a very light sleep from which the patient could easily be aroused", but this was in a group that were treated initially with "Librium" given intravenously, and Sussex (1960) recorded a case of a woman who "had felt 'very drowsy and strange' after the first dose", and noted drowsiness in other patients. Tobin *et alii* (1960) state that nearly 50% of 79 patients reported improvement in sleep, but that five patients said they had difficulty in falling asleep when taking the drug, and one patient had originally "complained of a tendency toward somnolence which was markedly improved" with "Librium". Vogt (1960) noted as a side effect "deeper sleep with tendency to fall asleep while sitting in a chair or resting". Fullerton and Bethell (1960) agree that drowsiness is a side effect of the drug and state it "is often greatest in the first few days of treatment and may disappear without reduction in dosage", whilst Denham (1960), Kagan (1960) and Vergano (1960) found the drug of value therapeutically with slight drowsiness as the

main side effect. Ingram and Timburg (1960), on the other hand, did not find the drug particularly useful and commented upon the frequency and severity of the side effects, noting drowsiness as one of some importance. Such features have been noted in a large number of our patients, the tendency to drowsiness being combated in most cases simply by a reduction in the dose. However, a number of the patients ceased taking the drug.

Two patients have recently been treated who demonstrated this side effect in a most dramatic manner. In short the effect was a period of dizziness followed by a sudden falling asleep. The circumstances of these two cases make the incidents worth recording.

Case I.

A male, aged 52 years, first came to the clinic as an out-patient, complaining of inability to control his drinking habits. He was an intelligent man with a very good position. His drinking tended to be periodic and was related to a build-up of tension, which in turn appeared to be related to a pronounced cyclothymic personality. He was treated with "Antabuse", supportive psychotherapy and "Librium" (20 mg. three times a day), and attended Alcoholics Anonymous. He found that whilst taking 60 mg. of "Librium" per day he was very tired and lethargic. The dose was therefore reduced to 40 mg. daily (20 mg. twice a day), with the desired effect that he was no longer tired and unable to work with reasonable efficiency.

He never worked on Saturdays and very rarely ate a great deal that day, having cups of tea and occasional snacks instead of more formal heavy meals. On one particular Saturday morning he took his two 10 mg. capsules of "Librium", had no breakfast, and two hours later began to feel ill. The episode began with a sense of dizziness, the room revolving round him in a number of different directions. He was not sweating. Soon afterwards he had a desire to defæcate, went to the lavatory and remained dizzy whilst sitting there. He shut his eyes and promptly fell asleep. The next moment he fell off the lavatory, sustaining a black eye. The fall caused him to awake, but he immediately fell asleep again, lying on the floor. He was awakened by his wife and, on leaving the lavatory, spent the next four or five hours asleep in bed. When he woke up he had no ill effects and he worked throughout the following week without any difficulty, although he was still taking "Librium" (40 mg. per day). The following Saturday a similar incident occurred. He had no breakfast, took his capsules and complained of dizziness and "an overpowering desire to sleep", a desire to defæcate and a mild abdominal colic. An attempt to defæcate was not successful, but ended in a less humiliating manner than on the previous occasion. He again slept off the effects and from then on spent an uneventful week. Since then the "Librium" has been stopped since the side effect was undesirable and the drug did not appear to be of any great value in treatment. Since ceasing to take the drug, the patient has had no further similar incidents or "overpowering desire to sleep".

Case II.

A male, aged 29 years, first came to the clinic complaining of phobic anxiety. He was almost unable to go out alone for more than a few moments and then only in a district he knew well. He was given "Librium" (60 mg. per day in divided doses after two weeks on half that dose). With the increase in dosage he complained of mild drowsiness, particularly when he was not busily occupied. On two occasions he fell asleep whilst out in a motor-car.

The first occasion was at 11 a.m., after he had taken his morning capsules at about 8.45 a.m. He suddenly went to sleep while waiting at some traffic lights. His companion woke him, and then took over the driving of the car after the lights had been passed. On reaching home he slept soundly for about three and a half hours. He had had a normal breakfast at about 8 a.m. The second occasion occurred at about 2.30 p.m. after he had taken the midday capsules at about 1.15 p.m. without lunch. He complained of vague dizziness of very slight degree and went out into the street to wash his car; after washing it, he got in the car to drive it into his driveway. Whilst driving the car in this way he suddenly fell asleep and hit a gate post. After this he again fell asleep, and was lifted out of the car and put to bed, where he slept off the effects.

Discussion.

It would seem probable that these side effects are produced by the drug acting on the reticular system and its hypothalamic connexions (Cohen, 1960). It would have been interesting to observe the effect of the drug on both these men under experimentally controlled conditions and to take electroencephalographic records and serial blood sugar level estimations, but both patients understandably refused to cooperate in such a plan.

This side effect has been described because, although perhaps rare, it is clearly of potential danger. At the present time there are few published trials with "Librium" that have been adequately controlled (Smith, 1960), so that due attention should be given to the prescribing of a drug that is still of uncertain worth, but that has at least one alarming side effect when given to an out-patient who may be particularly susceptible. Lawrence (1960) comments upon the low toxicity of the drug, but states, when considering the treatment of the out-patient with "Librium":

> In the out-patient these side-effects (drowsiness and mild ataxia) do not present a serious problem if a responsible relative or friend can be depended on to keep the patient under observation. In the out-patient where there is no one to assume this responsibility more conservative dosages should be employed.

As the two patients described exhibited the side effect on relatively small doses it would seem that all patients should be warned of possible lethargy (Walzer *et alii*, 1960) and drowsiness, particularly if they are driving a car.

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I should like to thank the Department of Mental Hygiene for permission to publish these two cases.

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Reviews.

Symposium on Glaucoma. Edited by William B. Clark, M.D., F.A.C.S., assisted by Joe M. Carmichael, M.S.J.; 1959. St. Louis: The C. V. Mosby Company. 9½" × 6¾", pp. 320. Price: £7 8s. 6d.

ONE of the main disadvantages of a published symposium is the uneven quality of the various contributors; it was a pleasure to read the present book, and to find that in this instance the contributions of all participants are of a uniformly high order.

The volume is dedicated to Paul Chandler. It is a tribute to American ophthalmology that such an excellent symposium on glaucoma could be produced without Chandler as a contributor. Every aspect of primary glaucoma has been dealt with by ophthalmologists who are recognized authorities on the particular aspect which they discuss. The list of authors includes Becker, Grant, Haas, Maumanee, Scheie, Swan, Dvorak-Theobald and Zimmerman.

The final chapter is a report of round-table discussions which were held at the end of each day's contribution, and this section alone makes the volume worth while from the point of view of the practising ophthalmologist.

The volume is beautifully produced, and the paper, type and diagrams are excellent. It can be highly recommended.

Drugs and Behavior. Edited by Leonard Uhr and James G. Miller; 1960. New York: John Wiley & Sons, Inc. 9" × 5¾", pp. 696, with illustrations. Price: $10.75.

THIS is one of an ever-growing collection of symposia on psychopharmacological topics. In contrast to some, which seem to be no more than hastily thrown together tape-recorded reports of conferences, there is evidence here of more careful preparation.

The work is divided into two main sections. The first, which deals with methodology and the chemical, biological and clinical background of the subject, is the better. Himwich has written an excellent chapter giving current views of the relationship of indole and catechol amines to mental symptoms. The anatomical site of action of these and of various therapeutic agents is well considered. At the same time, due warning is given of how far practice has outstripped theory.

After this, a chapter by Lehmann stresses the need for objectivity in evaluating the effects of psychoactive drugs. A plea is made for the use of the oldest and most complex of all psychiatric research instruments, the methodological and clinically experienced psychiatrist—a timely reminder to those whose gospel is that truth lies in the fervent application of increasingly complex batteries of psychological tests.

The second section, which is concerned with experimental procedures, may, according to one's point of view, be of lesser interest. Most of the experiments described lead only to cautious and indefinite conclusions. Some, at least, might be more appropriately placed within the confines of a journal than the covers of a textbook.

While it is of some value, the proper place for this book is probably in the stacks rather than as a personal library item.

Evaluation of Drug Therapy: Proceedings of the Symposium on Evaluation of Drug Therapy in Neurologic and Sensory Diseases Held at the University of Wisconsin, May, 1960. Edited by F. M. Forster. The University of Wisconsin Press; 1961. 8½" × 5", pp. 192. Price: $4.00.

THE purpose of this symposium was to bring together a group of workers—clinicians, pharmacologists and biostatisticians—who had as a common focus of interest the critical testing of drugs in the treatment of neurological and sensory disorders. This volume contains the papers presented during the two days of the meeting.

The first 88 pages deal rather broadly with some of the pharmacological problems involving new drugs, with the difficulties of testing drugs in the clinical trial, with the rôle of the food and drug administration in supervising the information set out by the manufacturers, and with the planning, design and evaluation of clinical trials in general. These sections are easy to read and interesting. They emphasize the vast amount of background work which goes into the discovery, trial and ultimate promotion of an effective pharmacological agent. It is pointed out that there are many ways in which a trial can go wrong, and in which much effort can be wasted. Some knowledge of the facts outlined in these pages would be valuable for any practising clinician who prescribes active therapeutic agents.

Reports from the panels set up to study a variety of specific disorders, such as epilepsy, the neuralgias, headache and glaucoma, make up the second half of the book. Definition and classification take up much of the time of the participants. The ideal disease for a clinical trial of therapy is said to be one with a uniform clinical picture, a predictable course, a specific diagnostic test and adequate frequency in the population. Multiple sclerosis, however, has few of these criteria, as it is a disease without known cause, with widely varied symptoms and signs, with an unpredictable course and without a specific diagnostic test. It is clear that this is a challenging but disheartening field for even the most gifted investigator.

This little book is worth perusal by those who are concerned with the scientific study of the action of drugs in disease.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Personality and Success in Marriage", by R. E. Morton; 1961. London: William Heinemann, Medical Books Ltd. 7¼" × 5", pp. 110. Price: 10s. net (English).

"Textbook of Medical Treatment", edited by Sir Derrick Dunlop, B.A. (Oxon.), M.D., F.R.C.P.Ed., F.R.C.P. Lond., Sir Stanley Davidson, B.A., M.D., Hon.M.D. (Oslo), F.R.C.P.Ed., F.R.C.P. Lond., and S. Alstead, C.B.E., M.D., F.R.C.P.Ed., F.R.C.P. Lond., F.R.F.P.S.; eighth edition, 1961. Edinburgh, London: E. & S. Livingstone Ltd. 9¼" × 6½", pp. 984. Price: 60s. net (English).

"A Short Manual of Venereal Diseases and Treponematosis", by R. C. L. Batchelor, M.A., M.B., Ch.B., D.P.H., F.R.C.S.E., F.R.C.P.E., and Marjorie Murrell, M.R.C.S., L.R.C.P., M.B., B.S., D.P.H., F.R.C.S.E.; second edition, 1961. Edinburgh, London: E. & S. Livingstone Ltd. 7¼" × 5", pp. 316, with illustrations. Price: 25s. net (English).

"Psychosomatic Aspects of Pædiatrics: Study Group of the Society for Psychosomatic Research held at The Royal College of Physicians", edited by R. Mackeith and J. Sandler; 1961. Oxford: Pergamon Press. 10" × 7", pp. 156. Price: 50s. net (English).

"Prosthetic Valves for Cardiac Surgery", edited by K. A. Merendino, M.D.; 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 6", pp. 586, with illustrations. Price: 66s. (English).

"Dermatology for Students", edited by R. O. Noojin, M.D.; 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 6", pp. 302, with illustrations. Price: 76s. (English).

"Diagnosis of Upper Gastrointestinal Hemorrhage", by Eddy D. Palmer, M.S., M.D., F.A.C.P.; 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 6", pp. 66, with illustrations. Price: 38s. (English).

"Kernicterus and its Importance in Cerebral Palsy", A Conference Presented by The American Academy for Cerebral Palsy, eleventh annual meeting, New Orleans, Louisiana; 1961. Springfield, Illinois: Charles C. Thomas; Oxford: Blackwell Scientific Publications. 9" × 6", pp. 306, with illustrations. Price: 70s. (English).

"Physiology of the Salivary Glands", by A. S. V. Burgen, M.D. (Lond.), M.R.C.P., and N. G. Emmelin, M.D. (Lond.); 1961; A monograph of the Physiological Society. London: Edward Arnold (Publishers) Ltd. 8½" × 5½", pp. 280, with illustrations. Price: 35s. net (English).

The Medical Journal of Australia

SATURDAY, SEPTEMBER 9, 1961.

COR PULMONALE.

A SMALL PROPORTION of patients suffering from chronic bronchitis and emphysema eventually develop congestive cardiac failure secondary to their chest disease (cor pulmonale). A considerable number of studies of this condition have been made, but most have been made on patients seen for the first time after cardiac failure has developed. Very few studies have been made in the same patients before and after the onset of failure, presumably because it is difficult for any one group of observers to witness the onset of failure in a large enough series of patients. Such a study could be carried out adequately only in a clinic where patients with chronic chest disease can be observed over a period of many years. M. M. Platts, J. D. S. Hammond and C. H. Stuart-Harris[1] have recently carried out such a survey over an eight-year period in a group of patients attending a clinic for chronic chest diseases at the Royal Hospital, Sheffield.

A total of 482 patients were seen, of whom 23 were observed and studied before and after the onset of heart failure. For purposes of comparison, 71 patients who did not develop cardiac failure were studied. Most of the patients had chronic bronchitis of varying severity, but some were suffering from bronchiectasis, asthma or pneumoconiosis. The object of the paper was to analyse the findings in order to determine which, if any, of the various measurements carried out were helpful as a prognostic indication in the development of cardiac failure. Special tests carried out included electrocardiography, radiography of the chest, tests of ventilatory function, oxygen saturation and carbon dioxide tension of the arterial blood and renal clearance as determined by renal plasma flow and glomerular filtration rate. Of the 23 patients who developed cardiac failure while under observation, 19 had chronic bronchitis and emphysema, three had bronchiestasis and one had asthma and emphysema. Only one patient was female. Nineteen patients died, and the clinical diagnosis of cor pulmonale was confirmed in the seven patients on whom autopsies were performed. The follow-up period prior to the onset of heart failure ranged from eight years to five months. For convenience of description, these 23 patients will be referred to subsequently as "cardiac" patients.

[1] *Quart. J. Med.*, 1960, 29:559 (October).

Ventilatory function, as determined by the vital capacity, indirect maximal breathing capacity, residual volume and mixing efficiency showed no significant difference in either group. In contrast, however, the carbon dioxide tension and arterial oxygen saturation were more often severely abnormal in the "cardiac" than in the other patients, and they were abnormal for long periods before the onset of heart failure. During heart failure, there was further lowering of the oxygen saturation and a further rise in the carbon dioxide tension. After recovery, the carbon dioxide tension and oxygen saturation were slightly more abnormal than before heart failure ensued.

There was no significant difference in the hæmoglobin concentration in the two groups, nor did the concentration rise in the "cardiac" group when heart failure developed. The cardio-thoracic ratio was normal in all the "cardiac" patients before the onset of failure. It increased simultaneously with the onset of failure, and tended to fall again when recovery took place. Electrocardiograms were performed on 17 of the 123 "cardiac" patients before the onset of failure. In 13 of these, various abnormalities were present. The commonest was a late *R* wave in lead aVR, found in 12 of the 17 patients. A right atrial *P* wave ("*P* pulmonale") was present in six. The *QRS* complex in lead VI was abnormal in seven, there being a delayed intrinsicoid deflection in three and an incomplete right bundle branch block pattern in four. Four patients showed inversion of the *T* wave in the right ventricular precordial leads. With the onset of heart failure, these changes increased in frequency. The group which did not develop heart failure showed a much lower incidence of these various changes, and none showed the delayed intrinsicoid deflection in lead VI.

Renal clearances were measured in 15 of the 23 "cardiac" patients. However, observations in the same patients before and after the onset of failure were made in only four cases. Clearances were estimated on 13 occasions in 11 patients from five to 61 months before the onset of heart failure. The glomerular filtration rate was greater than 100 ml. per minute on six occasions, and was moderately reduced on the other seven. The renal plasma flow was reduced in 11 of the 13 measurements. Hence the filtration fraction was higher than normal in eight of the 13 estimations. In the four patients studied before and after the onset of cardiac failure, there was a further reduction in glomerular filtration rate and renal plasma flow when failure occurred. The reduction in plasma flow was relatively greater than that in glomerular filtration, so that there was a further increase in the filtration fraction. During failure, a high filtration fraction was the commonest abnormality. These changes during failure are similar to those reported by other workers—C. E. Davies and J. A. Kilpatrick,[2] C. H. Stuart-Harris, J. Mackinnon, J. D. S. Hammond and W. D. Smith.[3] It thus appears that the increase in the filtration fraction, which characterizes the renal circulation in patients with all forms of heart failure, was present for many months before the development of œdema.

In conclusion, this study has confirmed the fact that there is no single reliable index of incipient heart failure

[2] *Clin. Sci.*, 1951, 10:53 (February).
[3] *Quart. J. Med.*, 1956, 25:389 (July).

in patients with chronic lung disease. The length of history, the clinical findings, and the disturbances in ventilatory function are of little avail in the assessment of the degree of involvement of the cardio-vascular system. The finding of a "*P* pulmonale" or a delayed intrinsicoid deflection in lead VI of the electrocardiogram, or of an increase in the filtration fraction, is probably indicative of serious involvement of the heart in patients with chronic lung disease. The alterations in the arterial blood gases are probably of more important prognostic significance. In the opinion of Platts and her colleagues, the presence of a raised carbon dioxide tension, or of unsaturation of the arterial blood with oxygen at rest, is the best evidence available at present of the likelihood of ultimate congestive cardiac failure. It is a matter of speculation, in the present state of our knowledge, whether it may be possible to prevent further deterioration in function at this stage of the disease by such measures as salt restriction, diuretic therapy and the use of digitalis.

Comments and Abstracts.

UNEXPECTED DEATH IN BRONCHIAL ASTHMA.

A RECENT, much-publicized case of death during an ashmatic attack makes the following report of topical interest. M. M. O'Brien and M. J. Ferguson,[1] of New York, discuss three cases of unexpected death in uncomplicated bronchial asthma, and describe what they believe to be an important prognostic triad observable in such cases. All three patients died in hospital, and their ages ranged from four to 44 years. The autopsy findings were almost identical in each case. When the chest was opened, the lungs were found to be voluminous, pale and distended. The pleural cavities were clear, and no adhesions were noted. When the bronchial tubes were opened, marked œdema of the mucosa was seen, and mucous plugs were found in many of the segmental and subsegmental bronchi. Microscopically there was no evidence of purulent infection. Examination of the hearts showed no evidence of hypertrophy. Clinical examination, radiography and electrocardiography had previously indicated that the hearts were normal in size, shape and function.

When the case notes were reviewed, the most striking clinical feature found was the consistent observation in each case that there had been an apparent auscultatory improvement shortly before death, in spite of increasing dyspnœa. In only one case had the clinician also noted that the breath sounds were decreased in volume, though O'Brien and Ferguson consider it probable that this change was present in the other cases as well; they point out that the attending physician is more likely to be impressed by the more obvious disappearance of wheezing. Their premonitory triad is therefore decrease in wheezing, increase in dyspnœa, and decrease in breath sounds. It should be noted that in one case the dyspnœa was thought to be due to psychosomatic overlay, and that in this and in one of the other cases the decreased wheezing on auscultation was interpreted as a sign of improvement, instead of as a sign of impending respiratory failure.

In their discussion of these cases, O'Brien and Ferguson postulate that the mechanism of respiratory failure is a progressive obliteration of the minor pulmonary radicles by mucous plugs, which leads to a decrease in the number of functioning alveoli, and results in compensatory dyspnœa. As the subsegmental and segmental bronchi are occluded, wheezing and breath sounds decrease. This process involves more and more of the lungs, the ventilatory capacity becomes severely reduced, and when a strategic bronchus is finally occluded, sudden asphyxia and death ensue. O'Brien and Ferguson emphasize that there were no signs of acute respiratory depression or of anaphylactic drug reaction in these cases. However, they suggest that the exhibition of pethidine and barbiturates may have contributed by weakening the cough reflex either directly or through their general sedative effect. They also note that oxygen was administered in all three cases, but without the addition of wetting agents. They point out that dry oxygen increases the viscosity and tenacity of bronchial secretions, and inhibits the tracheo-bronchial cilia. They therefore suggest that when the triad of decreased wheezing, increased dyspnœa and diminished breath sounds is recognized in the course of an asthmatic attack, the following points should be considered: (i) the patient should not be over-sedated; (ii) coughing should be encouraged and expectorants (e.g. potassium iodide) should be given; (iii) oxygen should be used only if necessary, and then only in conjunction with a wetting agent; (iv) adequate hydration should be ensured. If the patient does not improve, or deteriorates further, after such a programme has been instituted, O'Brien and Ferguson suggest that the induction of anæsthesia, preferably with ether, and the performance of bronchoscopy with diligent aspiration, may be life-saving.

[1] *Ann. intern. Med.*, 1960, 53: 1162 (December).

CHILD MORTALITY IN JAVA.

WITH the exception of Hong Kong, the island of Java has the highest density of population of any country in the world. The estimated figure for Java is 429 per square kilometre, which is to be compared with 241 for Japan and one for Australia. These stark figures are quoted by M. Timmer[1] in a book presenting the findings of a study of the factors associated with the infant and child mortality in the district of Jogjakarta, which lies in the middle of the south coast of Java.

Reported infant mortality lies between 17 and 162 per 1000 registered births in the different administrative subdistricts; the low figures are merely indications of incomplete registrations. Where a maternal and child health service has been operating for some time, the rate seems to be between 150 and 170 per 1000 births, which is judged by the author to be fairly close to the correct situation. The main causes of death in this age group are enteritis and prematurity; but underlying most deaths is severe malnutrition. It is interesting to note the distribution of deaths through the first year. In a well-advanced country like Australia, about 62% of the infantile deaths occur in the first week of life and 71% in the first month; in D.I. Jogjakarta the percentages are 12 and 55 respectively, emphasizing the impact of infection and malnutrition which take their toll towards the end of the first month of life.

The author devotes a number of chapters to descriptions of the clinical manifestations and epidemiological characteristics of diseases responsible for the majority of infant and child deaths; although of interest, these sections contribute nothing new to our knowledge of these conditions.

The most interesting chapters are those describing the agricultural, social and economic factors associated with the high child mortality. Outstanding among these are the population pressure on the limited arable land available and the relative lack of fertility of the soil in many areas; as a consequence, too many people live on marginal lands, where "mining" of the soil has led to progressive erosion and so to a further reduction in production.

Frequently the doctor in the out-patient clinic is tempted to label an ill, malnourished child as "neglect of the young child by the mother". The author points out that this is not true; children are highly regarded in Java, are

[1] "Child Mortality and Population Pressure in The D.I. Jogjakarta, Java, Indonesia: A Social-Medical Study", by Maarten Timmer; 1961. Rotterdam: "Bronder-Offset". 9¼" x 6¼", pp. 504, with figures. Price not stated.

positively appreciated at birth, and every child is supposed to bring its own happiness. The great majority of mothers operate comparatively well within the limited resources available to them. True it is that taboos which would restrict the child's intake of nutritious foods exist; but it is highly unlikely that the infants would get these if no taboo existed, because these foods are economically unavailable, so, as the author graphically expresses it, "the grapes are sour because they are hanging too high".

Malnourished children are found in the higher socio-economic group, but here ignorance is the main cause, and when this is corrected the prognosis for the child is good. When, however, ignorance and poverty are combined, the outcome is frequently fatal. This fact is emphasized in the marked difference between the birth weights of the infants of the poor and of those of the well-to-do.

The staple foods are rice and cassava supplemented with bananas, a few legumes and green vegetables and very occasionally meat. Breast feeding is prolonged, and supplementary feeding consists of thin rice porridge and bananas, so that the infant suffers from a deficiency of both calories and protein.

The author reviews possible remedies, such as increased production which is possible in the more fertile areas, industrialization which will require great capital expenditure, and transmigration which presents great problems in transportation. The impression gained is one of pessimism.

So far as Australian readers are concerned, this book serves to emphasize a most important fact, which many tend to exclude from their thoughts. To our north lies a most densely-populated island, where death rates due to starvation are high, where much of the soil is poor and where a marked increase in food production is doubtful and other solutions to the problem are not immediately visible. One is justified in asking how long this situation can continue.

THE MANAGEMENT OF CHEST INJURIES.

CHEST INJURIES have become sadly common in these days of frequent road and industrial accidents, so it behoves all of us to know how to manage them, especially in the immediate hours after the accident. Ideally these injuries should be treated in a centre where all equipment is available, with skilled personnel to use it. At times extensive damage can be overlooked unless it is especially sought. Unfortunately, too, there are often serious injuries to other parts of the body. The whole subject has been discussed recently by T. Holmes Sellors,[1] who comments: "The significant chest injury is one in which multiple fractures occur anteriorly and posteriorly to produce a functionally loose segment of the chest wall. . . . This 'flail' or 'stove-in' chest has the same effect as an open pneumothorax in producing paradoxical movement." There is also "the danger of retained blood and secretions in the bronchial tree". Anoxia may be almost silent; the "only sign may be a lilac tinge", and arterial oxygen and carbon dioxide estimation may be required for its detection. "The patient is often quiet and without cyanosis owing to shock that leads to a low cardiac output and to vasoconstriction."

In management three objectives, as outlined by N. Barrett,[2] are generally accepted, namely, (i) ensure that there is a clear airway and some temporary stabilization of a "flail" chest, (ii) ensure adequate ventilation and remove air and blood from the pleural cavity, (iii) make definite exploration and examination of the patient's injuries. Tracheostomy and positive pressure ventilation may be necessary later. Some of these points are brought out in two articles by H. M. Windsor and B. Dwyer.[3,4] Some of the special points they consider are the advantages of bilateral anterior thoracotomy for extensive mediastinal damage and/or involvement of both pleural cavities, and the ability to control paradoxical chest movements by full curarization and controlled respiration.

[1] *Thorax*, 1961, 16:1 (March).
[2] *Lancet*, 1960, 1:293 (February 6).
[3] *Thorax*, 1961, 16:3 (March).
[4] *Aust. & N.Z. J. Surg.*, 1960, 29:235 (February).

OSTEITIS PUBIS.

OSTEITIS PUBIS is a rare but troublesome complication of operations close to the pubis, and most commonly results from an infection in the retropubic space. The condition was originally described by Legueu and Rochet in 1923 as a sequel of suprapubic cystotomy. However, not much attention was paid to this extremely painful and disabling condition until after the introduction of retropubic prostatectomy. R. T. T. Warwick[1] discusses the pathogenesis and treatment of the condition in a paper in which he describes two cases of his own, one of which occurred as a complication of drainage of the retropubic space after iatrogenic rupture of the bladder, the other as a complication of retropubic prostatectomy. He states that insufficient attention has been paid to the synchondrosis itself in regard to both the pathogenesis and the treatment of this disease. His findings indicate that the synchondrosis is also infected in an established case of osteitis of the pubic bones, and that the relatively avascular fibrocartilage undergoes necrosis; this results in an interpubic abscess containing a soft-tissue sequestrum. Evacuation of this abscess with removal of the necrotic fibrocartilage gave dramatic relief in the cases of the two patients discussed, and greatly shortened the course of their illness. In each case culture of the debris from the interpubic space produced a growth of *Escherichia coli*, and histological examination showed inflammatory granulation tissue and necrotic fibrous tissue. Warwick states that on exposure of the symphysis by a suprapubic incision it is important to appreciate that the anterior and posterior interpubic ligaments may appear normal, and give no indication of the interpubic abscess and sequestrated synchondrosis they enclose. The infected synchondrosis is easily removed with dissecting forceps, but the operator should ensure that removal is complete by using gentle curettage.

In 1954, B. D. Stutter[2] suggested that the primary lesion in osteitis pubis was avascular necrosis of the interpubic disk resulting in impaired blood supply due to operation injury. The bony changes in the pubis were, he thought, secondary to this disk necrosis, and in the nature of avascular osteoporosis in most cases, rather than due to low grade infection or osteitis. Warwick, however, inclines to the view that the commoner lesion is simple infective necrosis of the interpubic disk and not avascular necrosis. It is immaterial whether the synchondrosis is infected initially in a given case, or by way of osteitis in the adjacent pubis, as each will result in the other if the disease becomes established. The bony changes are therefore symmetrically bilateral. The low-grade osteitis in the pubic and ischial rami takes up to one or two months to subside, but this resolution cannot begin until the interpubic abscess and soft tissue sequestrum have resolved, or been removed. It is interesting to note that Stutter, in spite of his different views on the pathogenesis of the condition, after surveying the unsatisfactory state of current methods of treatment, also suggested that excision of the necrotic interpubic disk might "reduce very considerably the duration of the disablement", though there is no evidence that he ever tried the operation himself. Warwick states that he has not found any previously reported case in which the synchondrosis was intentionally incised and completely enucleated at any early stage of the disease, before the surrounding structures become involved, though in some published case the symphysis was curetted as part of another local abscess, apparently with good results.

[1] *Brit. J. Urol.*, 1960, 32:464 (December).
[2] *Brit. J. Surg.*, 1954, 42:164 (September).

SHORTER ABSTRACTS.

MEDICINE.

CONTINUING INCAPACITY DESPITE "MEDICAL RECOVERY". M. H. Miller, *J. Amer. med. Ass.*, 1961, 176: 205-207 (April 22).

THE author discusses incapacity despite "medical recovery" and illustrates his remarks with the clinical notes of two patients. The physician not infrequently encounters patients who have achieved good medical or surgical recovery, but fail to return to a life of useful activity. Such patients show a continuing incapacity despite "medical recovery". This is especially likely to occur after illnesses that immobilize the patient and make him dependent upon others for his care. His relation to the physician and the physician's understanding and interest strongly influence the results. Discharge planning should begin at the time of the physician's first interview with the patient, and measures to keep the patient alert, stimulated, and self-confident should be instituted, especially if treatment involves long periods of immobilization. The physician must possess the flexibility in outlook and over-all breadth of perspective which allows him to be attuned to the patient's response to his illness, recovery period, and return to functioning. The author states that optional procedures which require long immobilization should be looked upon with increasing suspicion by all practitioners.

VALUE OF LONG-TERM ANTICOAGULANT THERAPY IN CORONARY DISEASE. A. B. Thomas *et alii*, *J. Amer. med. Ass.*, 1961, 176: 181-187 (April 22).

THE authors discuss the value of long-term anticoagulant therapy in coronary disease. The indications for discontinuance of treatment should be based on a quantitative comparison of the risk of continuance of treatment versus that of discontinuance, and it is primarily with this end in view that the present investigation is made. The study is a retrospective evaluation of the results of long-term anticoagulant therapy in a practice from 1948 to 1958. This study indicates that the advantage of anticoagulant treatment in a case of infarction continues and becomes continually greater over a period of at least several years. The underlying disease process is not arrested by anticoagulant therapy, but thrombo-embolic incidents in the course of the disease may be postponed during the maintenance of the therapy. The authors state that the incidence of thrombo-embolic episodes and recurrences of infarction has been shown to be significantly lower during uninterrupted anticoagulant therapy, as compared with the rate of recurrence after discontinuance. This difference increases in magnitude with time. The lower recurrence rate observed during treatment has been shown to obtain only as long as treatment is continued. Upon discontinuance, the advantage maintained by treatment is rapidly lost. A favourable effect on the mortality rate in patients treated with anticoagulants is suggested. In patients without evidence of diabetes or cardiac decompensation a very low death rate was observed throughout the duration of continuous therapy.

ANTICOAGULANT THERAPY IN INTERMITTENT CEREBRO-VASCULAR INSUFFICIENCY. R. J. Siekert *et alii*, *J. Amer. med. Ass.*, 1961, 176: 19-22 (April 8).

THE authors discuss anticoagulant therapy in intermittent cerebro-vascular insufficiency, and base their conclusions upon observation of 230 patients. These people experience attacks of neurological abnormalities which are the result of ischæmia of a portion of the brain. The attacks are brief, vary in frequency and severity, and occur over a period of months or years. Between the attacks the patient is normal. Duration of attacks only rarely exceeds one hour and usually is from 15 to 30 minutes. Attacks may occur only once a year, but in the majority they occur more frequently than once a month and sometimes as often as many times per day. The use of anticoagulant therapy has been associated with an immediate cessation of attacks. Of the 230 patients in this series, with intermittent focal cerebro-vascular insufficiency, all were followed up for periods varying from one to more than five years. A comparison was made as to the occurrence of cerebral infarction between those who received anticoagulant therapy and those who did not receive such treatment. Of 115 patients treated continuously, 4% had a cerebral infarction, and of 40 patients who did not receive this treatment, 40% had a cerebral infarction. Of 75 patients treated for a limited period of months, 32% had a cerebral infarction months or years after discontinuance of anticoagulant therapy. The occurrence of intracerebral hæmorrhage was the same in treated and untreated patients. It is concluded that this therapy is associated with a reduction in cerebral infarction in this particular category of cerebro-vascular disease. At least 83% of the treated patients, and at least 50% of the untreated patients did not have cerebral infarcts at the time of the follow-up.

THE CHANGING CLINICAL PICTURE OF DIGITALIS INTOXICATION. A. Soffer, *Arch. intern. Med.*, 1961, 107: 681-688 (May).

THE author discusses 24 cases of digitalis intoxication diagnosed during a twelve month period. Of these, nine were characterized electrocardiographically by atrio-ventricular dissociation with an atrio-ventricular nodal rate above 70 per minute. Recent emphasis on paroxysmal auricular tachycardia with block has obscured the fact that non-paroxysmal nodal tachycardia is a more frequent and valuable clue to digitalis intoxication. Four-fifths of these cases were induced by digoxin, demonstrating that a "safe" rapidly excreted purified glycoside such as digoxin can readily produce intoxication. Toxicity precipitated by initial digitalization occurred only if the administration was parenteral. The average age of patients in this series was 69·7 years. The ready precipitation of digitalis toxicity is a tragic characteristic of the aged and profoundly diseased myocardium, and attempts to administer average amounts of digitalis may lead to aggravation of the clinical condition. The use of potent potassium-losing diuretics often precipitated the appearance of toxicity, but determinations of the serum potassium level proved to be of little assistance in prophylaxis or diagnosis. The author concludes that there are many patients for whom potassium and digitalis therapy are contraindicated until other measures relieve to some extent the degree of congestive cardiac failure.

ACUTE BARBITURATE INTOXICATION. J. K. Dobos *et alii*, *J. Amer. med. Ass.*, 1961, 176: 268-272 (April 29).

THE authors present data from more than 400 cases of acute barbiturate intoxication, wherein evidence was sought of the possible effectiveness of analeptic drugs (such as caffeine, amphetamine or picrotoxin) in the treatment of this condition. The final comparison was limited to patients admitted to hospital in a condition of coma with laboratory evidence of barbiturates in urine, blood serum, or gastric washings. Of the 141 patients who satisfied these criteria, 77 received analeptics and 64 were treated supportively without analeptics. There were four deaths in the former group and two in the latter. Gastric lavage, as part of the routine therapy, was performed in nearly all cases. The duration of coma is not significantly altered by the use of analeptic drugs, and appears to vary directly with its depth. Intake of alcohol with a barbiturate contributes to the depth of coma but not to its duration. Not one death occurred in this series where the patient had ingested both alcohol and a barbiturate drug. The main complications of coma are bronchopneumonia and cardiac arrhythmias. The mortality rate of the group was 4·3%, which approximates to the over-all mortality rate of barbiturate-induced coma from all sources, irrespective of the regimen used. The authors conclude that patients treated with analeptics did no better than those treated supportively, in regard to duration of coma, number of complications, or rate of mortality. Until a simple, non-hazardous specific therapy has been devised, they consider that supportive measures alone, actively carried out, are sufficient for the treatment of most cases of acute barbiturate intoxication.

PULMONARY TOXOPLASMOSIS. M. Perrin-Fayolle and J.-P. Gatin, *Presse méd.*, 1960, 68: 1994-1997 (November 23).

THE authors present the results of an experimental investigation of pulmonary toxoplasmosis in mice. Under general (ether) anæsthesia, 50 mice received intranasally a known quantity of toxoplasma inoculum. Toxoplasmic invasion of the lung causes acute fatal disease, characterized by multiple interstitial foci of bronchopneumonia with diffuse hæmorrhagic alveolitis and œdematous and inflammatory thickening of all the alveolar septa. The peribronchial foci of pneumonia are produced by an infiltration of cells, chiefly lymphocytes. By the presence within them of numerous *Toxoplasma* organisms they are closely related to the inflammatory toxoplasmic nodules found in other organs. This lung disease, in which the cellular reaction is primarily of reticulo-endothelial type, can thus be classi-

fied with the parasitic reticulo-endothelioses. Two groups of 10 mice were treated with injections of sulphamethopyrazine and of spiramycin and trisulphazine. No cure was effected. It was not possible to observe the mode of healing and cicatrization of acute pulmonary toxoplasmosis of mice. Acquired pulmonary toxoplasmosis in man is seldom an isolated incident. It is most frequently observed during the major fatal septicæmic forms. The lung lesions are thus comparable with those in mice. One case of acquired human toxoplasmosis, an isolated lesion, presenting as a calcified nodule in the lung is reported; the lesion resolved spontaneously. This raises the question of infection through the respiratory tract.

COMPLETE HEART BLOCK TREATED WITH CORTICOTROPHIN AND CORTICOSTEROID. C. P. Aber and E. W. Jones, *Brit. Heart J.*, 1960, 22: 723-728 (November).

THE authors describe five cases of complete heart block in which the patients were treated with corticotrophin or corticosteroids. The rationale of this form of treatment is that heart block may be due to a reversible inflammatory reaction in the region of the atrio-ventricular node and bundle of His, rather than to permanent damage at this site, and that it may be possible to suppress this reaction and abolish the block with these inti-inflammatory drugs. The authors state that the natural course of complete heart block is so variable that no firm conclusions can be drawn from a small series of cases of diverse ætiology. It seems that this form of therapy may be valuable in overcoming persistent heart block when it is causing incapacitating Adams-Stokes attacks that have proved resistant to the more conventional therapeutic measures. It may be also the treatment of choice in those desperate cases of complete heart block occurring after a recent myocardial infarction or with severe myocardial damage whatever its ætiology, where the prognosis is uncertain and often grave.

ELECTROCARDIOGRAPHIC AND RADIOLOGICAL STUDIES IN HIATUS HERNIA. J. B. McGuinness and S. D. Scott Park, *Brit. Heart J.*, 1960, 22: 629-634 (November).

THE authors report an investigation by radiology and electrocardiography of 20 patients known to have hiatus hernia. After an initial electrocardiogram each patient swallowed a pint of thick barium emulsion and a further record was taken when the hernia was filled. In six patients electrocardiographic changes were produced by this procedure, involving Lead III in particular. These findings are discussed and it is concluded that in the presence of a full hiatus hernia alterations may occur in the electrocardiogram. These are attributed to positional changes and are considered likely to be confused with the changes of ischæmic heart disease.

PULSELESS DISEASE: A REPORT ON FIVE CASES. R. A. Caldwell and E. W. Skipper, *Brit. Heart J.*, 1961, 23: 53-65 (January).

THE authors state that pulseless disease (Takayashu's disease) is due to an obliterative process in the great branches of the aortic arch. They describe five cases of this rare condition. In two of these the disease proved fatal, and the necropsy and histological findings are given; in one patient the cause was considered to be a non-specific arteritis, in the other atheroma. Non-specific arteritis is the usual cause in young women. The possibility that congenital anomalies of the origins of the aortic arch branches may play a part is also discussed.

CHRONIC CONSTRICTIVE PERICARDITIS. P. Wood, *Amer. J. Cardiol.*, 1961, 7: 48-61 (January).

THE author presents an analysis of a series of 40 cases of constrictive pericarditis. A quarter of the patients were still in the active stage of the disease. A paradoxical pulse, a dominent and sharp *x* descent in the jugular pulse, normal rhythm, more than slight enlargement of the heart shadow and persistent hydrothorax were noted more commonly in the group with active pericarditis. Atrial fibrillation, a relatively small heart and pericardial calcification strongly indicated inactive lesions of long duration. An early third heart sound and a characteristic electrocardiographic pattern occurred with equal frequency in both groups. In the differential diagnosis the only serious difficulty was in distinguishing constrictive pericarditis from cardiomyopathy of clinically obscure origin. Factors indicating cardiomyopathy included a conspicuous left ventricular cardiac impulse, a third heart sound falling at its usual time, mitral or tricuspid regurgitation, bundle branch block, electrocardiographic changes denoting left ventricular hypertrophy or necrosis, and more than slight cardiac enlargement radiologically. The operative mortality was 11% in the 27 cases treated surgically. The results were good or excellent in 82%, and poor or indifferent in 7%. Reconstriction was not observed. Activity is no bar to successful surgical therapy; none of the seven patients with active pericarditis operated on in this series died.

GOUT IN CYANOTIC CONGENITAL HEART DISEASE. Jane Somerville, *Brit. Heart J.*, 1961, 23: 31-34 (January).

THE author describes nine patients with cyanotic heart disease, complicated by gout. Eight of the patients were males, the youngest being 18 years and the oldest 69. Fallot's tetralogy was present in four of the nine patients. The incidence of gout in cyanotic congenital heart disease is greater than in the general population. In all the patients studied the hæmoglobin value was over 130% and the serum uric acid level more than 6 mg. per 100 ml. at the time of the first attack. The development of gout is related to the degree of elevation of hæmoglobin and the age of the patient. The presence of renal disease may be responsible for its occurrence, particularly in patients under the age of 25 years.

PERICARDITIS: A TEN-YEAR SURVEY. D. C. Connolly and H. B. Burchell, *Amer. J. Cardiol.*, 1961, 7: 7-14 (January).

THE authors have reviewed the records of the Mayo Clinic during the ten years 1950 to 1959 and found that acute non-specific pericarditis was a relatively common diagnosis. The ætiological agent in many patients appeared to be a virus, and in a large group the process was related to a hypersensitivity state or an autoimmune process. Acute non-specific pericarditis is not always a benign disease; amongst the complications noted were pericardial effusion with tamponade, pericardial hæmorrhage, recurrences, the subsequent appearance of chronic constrictive pericarditis, and associated mild carditis. Adrenal steroid therapy may help dramatically in some patients, but steroid dependence and occasional hypercorticoadrenalism may be disturbing complications of this treatment.

ELECTROCARDIOGRAPHIC DIAGNOSIS OF MYOCARDIAL INFARCTION IN CASES OF COMPLETE LEFT BUNDLE BRANCH BLOCK. M. Besoáin-Santander and G. Gómez-Ebensperguer, *Amer. Heart J.*, 1960, 60: 886-897 (December).

THE authors have analysed the electrocardiograms of 13 patients with myocardial infarction and left bundle branch block. The diagnosis of infarction was based on autopsy reports, typical electrocardiographic changes in records without disturbance of conduction, and the presence of the classical clinical syndrome with evolutionary *ST-T* wave changes. The electrocardiographic changes were compared with those of 87 patients with complete left bundle branch block without myocardial infarction, and all the signs described in the literature as indicating myocardial infarction in the presence of left bundle branch block were studied. The conclusion is that only some changes in the *ST-T* waves (particularly if they are serial and not explained by the administration of digitalis) indicated with certainty a myocardial infarction. Most of the other signs described, especially those referring to the *QRS* complexes, were found in the absence of myocardial infarction and were therefore not diagnostic.

BILATERAL ADRENAL HÆMORRHAGE DURING ANTICOAGULANT THERAPY. A. Domart *et alii*, *Presse méd.*, 1961, 489-490 (March 4).

THE authors state that bilateral adrenal hæmorrhage due to the use of anticoagulants has only recently been described and is still rare. They have found six cases in the literature, and report one of their own. The condition generally manifests itself by collapse of sudden onset, the cause of which is discovered only at autopsy. In other cases the progress of the condition is slower, and hormone therapy may save the patient's life, but he is left with chronic adrenal insufficiency. These localized hæmorrhages, which are always bilateral, and which are not explained by simple overdosage with anticoagulants, seem to be due to capillary lesions and to be more frequently observed in subjects beyond middle age. The authors consider that the knowledge that this disaster can occur in association with anticoagulant therapy has an undoubted practical application: sudden collapse, in the absence of frank hæmorrhage, makes mandatory the immediate institution of hormone therapy, as in any case of acute adrenal insufficiency.

On The Periphery.

THE FIRST CONGOLESE DOCTOR.

THE World Health Organization has made available the following account of the first Congolese doctor.

There was not a single Congolese doctor when independence was achieved just over a year ago. Now, two citizens of the Congo have graduated from the medical faculty of the University of Lovanium, near Leopoldville, the first in history to do so. By 1965 there should be 18, and the World Health Organization has provided additional professors so that more students can be admitted in the medical faculty of that University each year. In addition, seven Congolese have completed the first year of medicine in universities in Europe on WHO fellowships, and the Organization has selected 60 "medical assistants" who are being given extra training in France to enable them to qualify as doctors as quickly as possible. The lack of medical practitioners nevertheless will long remain acute in the Congo. In 1958 there were 703 doctors, 82 pharmacists, 43 dentists and 11 biologists, all European, and most of them now have left the country. The deficiency is partly being met by external recruitment on an emergency basis. Ritchie Calder, the well-known British science writer, describes here his interview with the first Congolese doctor.

His name was Tshibamba Marcel. That was how he signed himself—surname first and first name second—a Congolese convention. To meet him I had to have an official appointment and a pass to get me past the guards, because he was Commissioner-General for Health for the Congo, a member of the *Collège des commissaires généraux*, a cabinet of students installed by Colonel Mobutu to run, temporarily, the affairs of the country. He received me in the ministerial office, flanked by his deputy commissioner and his *chef du cabinet*. But as we talked he forgot the cares (and perils) of an office he had never sought, and became once again an eager student whose greatest ambition was not political, but to become one of the first Congolese to qualify as a doctor.

Tshibamba Marcel was born in 1931 in a small village 30 kilometres from Luluabourg in Kasai Province. Thirty years seemed old for a medical student, but his history explained why. His father was an impoverished and illiterate cultivator, whose subsistence farming was so meagre that often his family went hungry. There were six children—four boys and two girls. Marcel was the fifth and the only one to receive education. He did not go to school until he was 10 years of age, when he went to the primary school in the village. It was a small school for small children who could not go long without food. That is what he said—hunger dominated their education. After three years Marcel was considered old enough to withstand the pangs of hunger. He was old enough to walk the 20 kilometres to Luluabourg and attend a better school there.

He told the story simply, how he went into the city and found a family whom he had never known, but who were prepared to take in the wandering pupil and share their poverty with him. About once every three weeks he would walk 40 kilometres to his village and back, to see his family, and if there was any food to spare to bring it back to his benefactors. Sometimes his father or brothers would come into Luluabourg bringing manioc and meat. But sometimes there was no food. Even his studies were determined by his stomach. Sometimes he was so hungry he could not go to school.

In 1945, he finished his primary stage and went to a Catholic residential school at Kabwe, 40 kilometres outside Luluabourg. The education was free. He made an application, and the tests on which he was accepted were of "merit", "conduct" and "concentration". He was admitted for a year on probation and remained until 1951. His special subject was poetry.

Without having fully finished his secondary school education, he went on to Kisantu, one of the training colleges for medical assistants, the highest medical category the Congolese could attain before independence. At this school he had two preparatory years before he started his main course. At the end of his second year, he had a stroke of good fortune. The University of Louvain, in Belgium, decided to establish a daughter university, Lovanium, in the Congo. At the outset it was doubtful whether there would be a medical faculty or whether, if there was, Africans would be admitted to it. The University of Louvain, however, insisted, against all opposition, that Lovanium should be a comprehensive university including faculties of theology, law, medicine, philosophy and pedagogy, as well as a polytechnic institute and an institute of agronomy, and the faculties were to be open to Africans.

In April, 1953, the foundations of the university were laid on the plateau of Mont Amba, in the hills above Leopoldville. (Today the university, with many of its buildings still to be completed, forms architecturally and conceptually a magnificent academic complex, with its teaching hospital and medical school and a nuclear reactor, the only one in Africa, for the production of radioisotopes for medical research and treatment.) In 1954, the first candidates were admitted to the medical faculty.

Tshibamba Marcel was one of the five Africans to be accepted. Three of them fell by the wayside; he, however, managed to pass his final examinations in July of this year, in spite of the difficulties of study in this last eventful year and the cares of office. Tshibamba Marcel has now become Dr. Marcel Tshibamba.

Congresses.

THE THIRD WORLD CONGRESS OF PSYCHIATRY.

IT is significant that the recently held (June 4 to 10, 1961) Third World Congress of Psychiatry in Montreal, Canada, had an attendance of about 3000 psychiatrists from 62 countries. Already from various popular reports it is known that the Congress was extremely well organized and not less well attended. (It is also significant that a few weeks ago, in New South Wales, the Minister for Health, Mr. W. F. Sheahan, introduced a number of modern improvements in the mental health programme, which was discussed in a leading article in this Journal on August 12, 1961).

A very important aspect of this World Congress of general interest is that, before the end of the proceedings, the members thought fit to found a World Psychiatric Association, to replace the International Psychiatric Congress. The new organization, which elected as its first president Dr. D. E. Cameron, of Montreal, was acclaimed enthusiastically by all members in an impressive meeting. Support was given by all delegates from all countries including Eastern Europe and Russia. It will not be too long before we shall hear more of this newly-founded organization, which, it is to be assumed, will coordinate its function with the World Health Organization and UNESCO and with each member country concerned. Preventive psychiatry, teaching psychiatry, transcultural aspects, clinical research, care of the aged and a number of other specialized fields such as forensic psychiatry, are vital not only to psychiatrists, but to the medical profession as a whole. Everybody of goodwill, psychiatrist or not, will support the new organization wholeheartedly, because it is aimed at the benefit of the mentally sick, independent of colour, creed or race.

One of the interesting highlights of the World Congress was the setting of the Congress in the Queen Elizabeth II Hotel recently built in Montreal. The so-called Congressional floor in this hotel contains a number of large auditoria, some of which are interconnected and were large enough to hold meetings with 3000 delegates. On the same floor there were about 20 smaller conference rooms and auditoria where continuous sessions and panel meetings were held. (Somebody once remarked sarcastically that congresses of this type are rather like a five-stage circus, where at the same time on various stages performances are given and

one cannot concentrate on all five and needs must make a choice. This may be true.)

A glance through the programme of the Congress indicates that a number of important papers were given. One unforgettable experience for most of the members was an evening when three Nobel prizewinners discussed "Scientific Creativity". The three in question were A. Szent-Gyorgyi (discoverer of vitamin C), Lord Adrian (neuro-physiologist, from England) and L. Pauling (Nobel prizewinner for laws concerning the molecule). These three lectures were delivered with great sense of humour and left pleasant memories. They were given in the Sir Arthur Curry Memorial Gymnasium of the McGill University. Although each of the speakers was limited to half an hour, they were able to convey to the very large audience their own experiences before they made their discoveries. The most down-to-earth in his talk was Lord Adrian; but, as the chairman of the meeting, the Dean of McGill University, Dr. D. L. Thomson, remarked, even Lord Adrian would have to say at the end of the meeting that "it was a jolly good show".

It should be recorded that Australia (especially New South Wales) was well represented. Two papers were read by Australians; Dr. Ainslie Meares of Melbourne discussed "Hypnosis, Yoga and the Pseudo-Trance States", and Dr. Oscar R. Schmalzbach read a paper entitled "Litigation Neurosis" and showed a film entitled "Conversion Hysteria Successfully Treated by Narcoanalysis".

The three opening addresses of the Congress were given by Professor Rumke of Utrecht on "Phenomenology", Professor H. C. Masserman of Chicago, who discussed "Experimental Psychiatry", and Professor H. A. Ey of Paris, who read a paper entitled "Theory". These three leading themes were delivered to the general session, and probably the most impressive was Professor Rumke. A number of other interesting and important subjects were discussed, some by the Russian psychiatrists. The Congress was also attended by members from various Eastern European countries.

One must agree that the Third World Psychiatric Congress will not only help the mentally ill, but will stimulate through personal contacts of the individual members and national groups happier human relationships. This should help to diminish and maybe to remove the international tension and anxiety and fear in everyday life, and bring about a better understanding of human behaviour, creating the basis for the happiness of all men.

Special Correspondence.

PARIS LETTER: MARCH-APRIL, 1961.

[FROM OUR SPECIAL CORRESPONDENT.]

A Theory of the Ætiological Factors in Bronchial Cancer.

THE various ætiological factors involved in the genesis of bronchial cancer have been repeated for the purposes of analysis.[1] Actually, when a particular case is considered, the various factors are found to be present in the one patient. Are we now in a position to plan a preventive campaign, since we know some at least of the causes?

To prevent the use of tobacco is for all practical purposes impossible, and many doctors, convinced of its dangers, continue smoking, even in the corridors during cancer congresses. Can we at least minimize the danger? Filters are useless. The choice of cigarette papers and their chemical treatment are important. The elimination of the precursors of carcinogens is impossible, and Neukomm concludes a recent article with the following words: "The anti-cancer cigarette, like the anti-benzopyrene cigarette, is a myth."

Improvement of working conditions in industry and protection against dust and smoke may decrease the risks.

The greatest problem is undoubtedly that of atmospheric pollution. The separation of housing districts from industrial areas; the elimination of factory smoke, as has been done at Pittsburgh (with a 65% reduction in air pollution); improvement of the fuel at present used in motor vehicles and especially in heavy Diesel motor vehicles, with a resulting improvement in exhaust gases; the encouragement of central heating in the cities (domestic fires in Paris produce 50% of the pollution); all these measures do not amount to a Utopia, but are possible both technically and financially. They would be good investments, and might, after a number of years, succeed in breaking the rising curve of bronchial cancer, and finally reduce the incidence.

[1] Fauvet, J. (1961), *Rev. praticien*, 11: 849 (March 11).

The Anthropological Problem of Thalassæmia.

The genotypic erythrocyte disorders at present can be divided into two groups—first, those found everywhere, and secondly, those particularly affecting certain racial groups (for example, thalassæmia and the hæmoglobinoses).[2] The existence of these abnormalities particularly affecting certain nations or certain races was bound to attract the attention of the anthropologists. Clinically, there are two distinct forms—Cooley's anæmia, which is a homozygote type, and thalassæmia minor of Rietti-Greppi-Micheli and the minimal thalassæmias, which are heterozygote in type. Thalassæmia occurs only in a restricted zone of the world, but this zone is enormous. Some non-racial explanations have been put forward. The importance of malarial infestation, of under-nourishment and of localized mutations has been advanced. Other writers have been led to think that the disease may have originated in one of the races coming into this enormous group, and that it may have spread progressively among the neighbouring peoples; this is observed to be occurring at present in America and Australia. Many have held that the disease was of Greek origin; but Greece, historically and politically a single unit, presents no independent racial unit. Other workers have thought that the disease was tied up with an ancient Mediterranean race; but recent work has failed to provide proof of the existence of this race, and the peoples of the islands, such as Sardinia, continue to present many problems. Still other writers have favoured a Mongol origin of the disease, since Asiatic foci have been found. Brumpt has even suggested that it should be called "sinæmia", since it is found in Bengal, in Sindh and in the Punjab, as well as in South-East Asia. It would then have progressed from the Mongolian steppes to the Middle East and the Near East by the same routes as the major migrations, and from there spread over all the Mediterranean basin. Finally, other workers have felt obliged to find some link in the vast group affected by the disease, and hold that it attacks the most southerly of the white races, the "brown dolichocephalic" group. In the centre of this group are found the Mediterranean race, the Southern Oriental race and the Indo-Afghan race. It is obvious that the anthropological interpretation of the problem of thalassæmia is incomplete, and that much more work will be necessary to solve it.

Such work should be undertaken on a world-wide scale, so that information may be obtained on the regions where our data are incomplete or entirely lacking. It is easy to see how valuable it would be to have the facts concerning South-East Asia and Central Asia. The investigations should cover both biology and anthropology, without prejudgement of any correlations that may be found. By this means perhaps the problem might be cleared up, by defining a new anthropological characteristic and by marking its genetic aspect on the population map.

The Pain of Mitral Stenosis.

Anginal pain has been studied in 1000 subjects suffering from mitral stenosis.[3] The following were eliminated from the group: all patients whose pain appeared first after the age of 50 years; women who had undergone hysterectomy; patients showing signs of aortic stenosis, hypertension or arterial degeneration, or of hypothyroidism or hyperthyroidism. Of the 732 cases to which these criteria applied, the authors picked out 28 in which angina pectoris seemed directly related to valvular disease. Under these conditions, its total incidence is stated to be 3·8%.

The only unusual features were the young age of the patients (an average of 34 years) and the frequent association of attacks of œdema following exertion.

The absence of coronary atherosclerotic lesions in the proven cases, and the electrocardiographic signs of ischæmia found at rest and during exertion in 26 out of 35 cases, suggest that this symptom is probably connected with functional coronary insufficiency resulting, on the one hand, from an increase in pulmonary artery resistance, and on the other, from an inadequate increase in cardiac output during exertion.

[2] Gomila, J. (1961), *Concours méd.*, 83: 1795 (March 23).
[3] Chiche, P., *et alii* (1961), *Sem. Hôp.*, 37: 1131 (April 2).

In the absence of aortic stenosis and of signs of arterial degeneration, the discovery of angina in a subject of mitral stenosis argues in favour of a tight stenosis.

Psychiatric Problems in a General Medical Service.

After considerable soul-searching, L. Justin-Besançon, H. Pequignot and F. de Paillerets[4] have come up with the idea that a general hospital, if it is truly to adapt itself to current psychiatric progress, must itself progress by revising the conception of its functions, its discipline and its location. Amongst the most frequently found deficiencies the authors note lack of knowledge of the abnormal characteristics of suicidal conduct, the lack of psychotherapeutic attitudes or of psychiatric diagnosis in the face of cœnæsthopathy or of hypochondriasis regarded as "functional", the lack of suitable measures for dealing with intoxicated alcoholics, the abandonment of inquiries into character, and finally, the fact that internal conflicts in the hospital service are resolved by disciplinary decisions, without any attention to the fact that such conflicts are often connected with disturbed psychological conditions.

The sum total of these psychiatric problems affecting the major general medical services leads the authors to plead for a large staff of psychiatrists and the generous provision of psychiatric services, so that there may be created in these medical departments a psychotherapeutic and educational atmosphere; the value of this would by no means be limited to psychopaths alone. At all events, the numerical importance of the problem (certainly more than two patients in 10, possibly more than three), and its appearance and development during the neuroleptic era and at a time when certain other disease entities are decreasing, demand of the clinician more and more a psychiatric approach to his work and some accurate knowledge in this field, if he does not wish to place himself under a severe handicap.

Can the Classical Treatment of Apnœa in Premature Infants be Improved?

Long periods of respiratory arrest with rapidly developing cyanosis are frequent accidents affecting premature infants in the first days of life, and even in some instances during the first weeks.[5] The classical treatment is that of freeing the respiratory passages from obstruction, but failures are not infrequent.

Autopsy does not always reveal cerebral or pulmonary lesions adequate to explain this outcome, and Alison holds that in this case functional disorders may be at fault. In 14 cases of severe apnœa, he has given into a scalp or peripheral vein infusions of the following solution (the figures are per kilogram of body weight and per 24 hours): plasma, 25 to 50 ml.; Ringer's solution, 35 ml.; serum with 50% glucose solution, 15 ml.; "Terramycin", 20 mg.; "Spirogene" (ammonium phthalamate), 1 ml. The total dose is 75 to 100 ml. per kilogram per day.

Of the 14 patients, 10 survived. Undoubtedly, all these babies would not have died if they had not been perfused, but we know the gravity of subintrant apnœa, the mortality rate of which is about 50%. It seems that the results achieved should encourage the use of this form of treatment.

Mortality in Western Europe Since 1900.

From 1900 to 1950, the countries of Western Europe have seen a considerable decrease in their mortality.[6] The absolute gain (almost 20 years more of life on the average) has been greater than that achieved in the previous century. It is unlikely that so much progress will be achieved in the next half century. The superiority of the Scandinavian countries from this point of view, which was pronounced in 1900, has been maintained; however, a trend towards regrouping is noticeable and seems likely to be accentuated. A number of studies have shown that mortality defined, for example, by average life span depends less on economic status than on the medico-social set-up in the most general sense of the term, at least whenever the standard of living has exceeded a certain minimal threshold. It is shown clearly by the work of J. Chasteland that the classification of countries in accordance with the standard of living will differ greatly from the classification based on the average life span.

The masculinity of the mortality rates continues to increase at all ages over the whole period 1900-1950, the increase being particularly noticeable for the age groups from 40 to 60 years. Two European countries have exceptionally high mortality among men for 1950: Finland for all ages except from birth to one year, and France from the beginning of adult life. Over the whole duration of human life, the high masculinity of the mortality rates has increased least in Scandinavia, and especially in the Netherlands. Thus, this phenomenon cannot be linked only with improvement of standards of living, since the countries in which these are particularly high, like Denmark and the Netherlands, have less masculinity of mortality than poor countries like Spain and Italy. In France, the connexion between this phenomenon and alcoholism has been shown by a number of studies, for example, those of S. Ledermann.

[4] *Sem. Hôp.*, 1961, 37: 831 (March 8).

[5] Alison, F., and Courtois, M. (1961), *Sem. Hôp., Ann. Pédiat.*, 37: 1264 (April 21).

[6] Chasteland, J.-C. (1961), *Concours méd.*, 83: 1645 (March 18).

Out of the Past.

WOMEN DOCTORS.[1]

[From the *Australasian Medical Gazette*, March 20, 1903.]

A WEEK or two ago representatives from the Sydney Women's Progressive Association waited upon Dr. Ashburton Thompson, President of the Board of Health, and expressed a strong wish that women doctors should be appointed to all public hospitals. After listening to the arguments advanced in support of the request, Dr. Thompson pointed out that he had appointed a woman doctor to the Coast Hospital. He added that in general his sympathies were with the views expressed by the deputation, but directed attention to the fact that he had no control over any of the hospitals other than those referred to. It is somewhat quaint to read alongside of this "forward movement" the report of such a discussion as has taken place at the German Medical Congress on the admission of women to practise in that country. It was unanimously decided that medicine would "suffer in the public esteem" if it were open to women. Thence it followed that it should not be so opened. The delegates also protested against "any attempts to make the conditions of study of entrance less onerous to suit female capacity"!

[1] From the original in the Mitchell Library, Sydney.

Obituary.

JOSEPH RINGLAND ANDERSON.

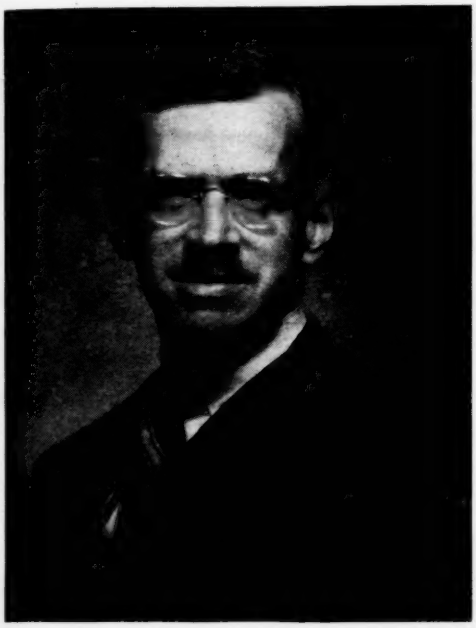
WE are indebted to DR. T. A'B. TRAVERS for the following account of the career of the late Dr. J. Ringland Anderson.

When Joseph Ringland Anderson died on May 14, 1961, Australia lost one of its leading figures in ophthalmology. Dr. Anderson was the son of the Reverend J. R. Anderson, and was educated at Scotch College, Melbourne. He graduated in 1916, and immediately enlisted with the Australian Imperial Force and served in France with the 45th Battalion. He was awarded the Military Cross for conspicuous bravery in pushing his regimental aid post forward under heavy fire. After the war he went to Britain for post-graduate study, and took the F.R.C.S. in Edinburgh in 1919. He returned to Melbourne, and soon earned a reputation as an ophthalmic surgeon of exceptional skill. He was appointed ophthalmic surgeon to the Alfred Hospital and became an acknowledged leader in his profession. In spite of his enormous practice he wrote several books. "Detachment of the Retina" was published in 1931, and "Hydrophthalmos" and the second edition of his book on "Ocular Vertical Deviations and the Treatment of Nystagmus" were published in 1959. He played an important part in the founding of the Ophthalmological Society of Australia (B.M.A.) and was a former president. He was also very active in the foundation of the Ophthalmic Research Institute of Australia. After the second World War he and his wife played a prominent part in trying to help orphan children in Japan. Dr. Anderson is survived by his wife and two daughters.

I should now like to add a personal tribute. I first met Dr. Anderson in 1927, when I had the good fortune to be his house surgeon. I say "good fortune", for it was he who first introduced me to the fascinations of ophthalmology. After I had worked for him, I knew that the

study and pursuit of eye diseases would be my mode of life also. In those days, and perhaps for the 20 years following, he was at the height of his powers. He had immense energy and never seemed to tire. He was enthusiastic about new work, and was always ready and willing to discuss interesting cases with his juniors. After I had done my post-graduate work in England I again worked with him as a clinical assistant in the Eye Department at the Alfred Hospital. I could now appreciate his qualities more accurately, and could realize how really good he was. His knowledge was immense, and he was always adding to it. He had a most beautiful pair of hands, and I think he was one of the best operators I have ever seen. No wonder he had an enormous practice; and yet, in spite of this he had time to write books and papers, well recognized to be in the first class.

I feel I should mention his extreme modesty, and his gentleness of manner. He was a deeply religious man, and perhaps this gave him a sweetness of approach that endeared him to his patients and gave him their trust.



In the second World War he was one of the consultants in ophthalmology to the Royal Australian Air Force, and it was while travelling from one camp to another that he was involved in a bad motor smash. He was admitted to Heidelberg Hospital and I, for one, did not expect him to survive; but with uncomplaining determination he slowly recovered and recommenced work. Before the accident he had been a fine athletic figure, but now he walked with a limp and his right forearm was badly damaged—one would think a crippling blow for such delicate work as eye surgery. But not for Joe Anderson! He went about his work with such a firm resolution that one hardly noticed any change in him, his sweetness of manner unchanged and his eyes still sparkling with enthusiasm.

But I think the accident did take its toll, for after the war he relaxed his private practice and became actively engaged in the affairs of the Ophthalmological Society. He was very keen on the exchange of overseas visitors, and represented Australia with great distinction at an International Congress of Ophthalmology in London in 1950. In his last years he dedicated himself to the establishment of a chair of ophthalmology in Melbourne, and by his own efforts much money was raised for this purpose. It must have been a great joy for him when the first professor was appointed only a short time before he died.

One would think that with all these things to fill his mind there would have been no room for anything else; but far from it. He was greatly interested in gardening, and had a beautiful garden at his home. He loved art, and was a follower of the classical ballet. In his later years he became an enthusiastic admirer of things Japanese, and with his ardent nature he strove to better the relations between Japan and Australia, so badly damaged by war.

A melanoma was removed from his finger and proved to be malignant. A year passed, and he had secondary deposits in his chest; but not a word of complaint. He persisted with grim determination and was seeing patients only a few weeks before his death. With his gentle smile he said that he only had a cold. And now he is gone, and we are all the poorer—and perhaps the richer, too, for his example of Christian virtues, honesty, gentleness and unshakeable courage.

SIR NORMAN GREGG writes: It would be impossible to overstate how deeply ophthalmology in Australia is indebted to Dr. J. Ringland Anderson. His contributions to the literature, whether in his several books, monographs and papers, were all of outstanding merit, and have won approval not only in Australia, but also abroad, especially in the United Kingdom and in the United States of America. In this way he played a leading rôle in obtaining international recognition for Australian ophthalmology.

In his own country we will ever be indebted to him for the part he played in the formation and establishment of the Ophthalmological Society of Australia (B.M.A.) and later of the Ophthalmic Research Institute. Always devoted to the advancement of the specialty, he recognized the need for the formation of an Australian society as an essential step to achieve progress. One of the original small group who set out to form this Society, he was also, I feel, its most ardent and enthusiastic advocate. When some may have thought that the size of the country and the long distances separating the capital cities would militate against a successful launching and running of such a combined society, his confidence in the scheme and his enthusiasm brushed aside all argument against it, and slowly but surely won over potential opponents into active and keen supporters. A foundation member of the Society, later a president, a member of various subcommittees, he was at all times one of its foremost and most influential personalities.

He again played a leading rôle in the formation of the Ophthalmic Research Institute of Australia. A signatory to the Articles, he was a foundation member of the Board, and member of the Research Committee from the inception up to the time of his death. His enthusiasm for the Institute was unbounded, and by his ability to infect others he was instrumental in obtaining substantial sums for the Institute and also for the establishment of a chair of ophthalmology at the University of Melbourne. It would be a fitting tribute if this chair could bear his name.

In the practice of his profession, he justly became recognized as one of the outstanding ophthalmologists of Australia. Ringland Anderson was a man of great natural ability and mental capacity, who strove at all times to add to and broaden his knowledge. He had, too, the ability to pass on to others the fruits of his knowledge and experience. He was always ready to help if his advice was sought, but he would never attempt to force his opinion on others. There was, I believe, a natural shyness and nervousness in his character, so that on a first or casual acquaintance he may have appeared unduly reserved. If that was a first impression, further acquaintance readily proved it to be false as one became more and more aware of the man himself—of his warmth, his honesty and sincerity, and at the same time, his extreme modesty.

By his friends "Joe" Anderson will always be remembered for his courage—the kind of courage with which he fought back and onwards after his very severe accident, and with which he faced his final illness. We think, too, of his charm of manner, his graciousness and his softness of speech.

Yet Joe Anderson was not a "yes man"—far from it. The opinions he held he was prepared to voice in his quiet yet forceful way, because he sincerely believed them to be sound and correct. He was, however, ever ready to listen to the other man's opinions, believing that they were formed just as sincerely. If in committee the contrary opinion was adopted, he would graciously accept the verdict without any feeling of resentment and would loyally abide by it.

He was a truly great man, who set a standard of service and achievement which we should strive to emulate. To his widow and daughters we offer our very sincere sympathy.

AIR VICE-MARSHAL E. A. DALEY, C.B.E., Q.H.P., writes: The fine qualities and character of Dr. J. Ringland Anderson

and his great work in ophthalmology can be commented on best by those who were more intimately associated with him in student, post-graduate and specialist practice. However, there is a special facet of his work which not only brings out clearly his fine and inherent sense of duty towards his fellow men and his country, but highlights his ingenuity and originality. Although he served as a medical officer in the 1914-1918 War and was awarded the Military Cross, on the outbreak of the 1939-1945 War he was one of the band of ophthalmologists who offered their services to the Royal Australian Air Force in the national cause in the early days. Apart from the importance of ophthalmology in its relation to flying as such, J. R. Anderson had been very impressed with the work done by Air Commodore (later Air Marshal) Sir Philip Livingston on orthoptics. He felt that along these lines, although the matter was controversial, there was a strong case to implement such training and investigation amongst the large number of air-crew trainees coming forward under the Empire Air-Training Scheme. The chance of helping border-line subjects and those with post-traumatic ocular muscle malfunctioning with a view to training them to see more confidently, and possibly to learn to fly more easily, stimulated Anderson into raising the matter with the Director-General of Medical Services of the Royal Australian Air Force, Air Vice-Marshal T. E. V. Hurley. In spite of professional differences of opinion with regard to the relationship of heterophoria, depth perception ability and skill in learning to land aircraft, Hurley agreed to Anderson's plan to introduce testing and orthoptic training amongst Australian air crew at the various initial training schools in Australia. Anderson supervised this work, and organized teams of orthoptists from civil life to aid its implementation. He assisted in the training of medical officers at these schools, and extended it to selected members of the nursing service. Special forms and records were compiled, and the scheme worked actively to the end of the war. In this work he became an enthusiastic driving force, and many air-crew trainees expressed their thanks and admiration for the training given them. Controversial or otherwise, one often heard the phrase: "I see more confidently when flying."

It was in travelling from Benalla with his orthoptic team that he sustained severe injuries in a motor-car accident, which involved him in some months in the Royal Australian Air Force Hospital, Heidelberg. Nevertheless, the work continued, and his only regret in connexion with it was the rapid loss of personnel on demobilization, who might have been able statistically and in other ways to collate the data obtained.

He studied the problem of "crossed dominance" in air crew, and worked on depth-perception apparatus, in his endeavour to improve subjects with good potential otherwise, but presenting bad heterophoria risks. In many other ways, in addition to his hospital and private work, he devoted time and energy to the ophthalmic problems of the Services. For example, the tremendous glare in Rabaul Harbour at night from the enormous mirrors and searchlights of the Japanese was disorientating air crew in attacking the area. Few people knew that behind the screen in his Collins-Street surgery was a model of Rabaul Harbour on which he was working in an endeavour to solve the glare problem. Dr. (now Sir John) Eccles also worked with him in some of his research. His generous expenditure of thought and energy, coupled with high professional approaches in these fields, undoubtedly was a unique and valuable contribution to the medical profession, the Royal Australian Air Force and the nation. Since these times he has still given of knowledge, experience and enthusiasm in helping to solve ophthalmic problems in the jet era. The Royal Australian Air Force generally and hundreds of ex-air crew of the war and since will mourn his passing, and remember the kindly, enthusiastic worker on their behalf.

JAMES MACRAE YEATES.

WE are indebted to DR. F. H. MILLS for the following account of the career of the late Dr. J. M. Yeates.

James Macrae Yeates was born at Toowoomba, Queensland, on November 2, 1909, into a notable family. He was one of five brothers, all of whom have had interesting and exceptional careers, both in the military sphere and in private life, and of whom two others are in the medical profession. His father was a member of the Legislative Assembly in Queensland, and his strong character played a large part in developing the quiet strength of James Yeates' personality. Another early influence was that doyen of headmasters, the late G. P. Barbour, of Toowoomba Grammar School, and James Yeates greatly enjoyed the life there. He became a good "all rounder", and was prominent in school sports, was well up in examinations and became a prefect during his last two years at school. From school he entered the Faculty of Medicine at the University of Sydney, from which he graduated M.B., B.S., with honours in 1934. He was appointed a resident medical officer to Sydney Hospital in 1935 and later surgical registrar in 1936. At this stage he had decided on general surgery as a career, and this took him to London in 1937 on the well-trodden path that leads up the stairway of the Royal College of Surgeons. London, in those days just before the war, and in the early war years, was a fascinating place, more so than it has ever afterwards appeared. It

was the centre of the world, where a succession of international crises had their biggest impact. James Yeates revelled in this atmosphere, became a frequent visitor to the debates in the Houses of Parliament and saw at first hand the growing menace of the rearmament of Germany. He was one of the many young doctors called up for emergency duty at the time of the Munich episode, and had a brief taste of the military life in which he later became so distinguished. Here also he had the opportunity to indulge his talent for literature and music. He was deeply musical and spent a great deal of time following this interest. During that time he was house surgeon to Professor Grey Turner at the British Postgraduate Medical School at Hammersmith, and he gained his F.R.C.S. (England) in February, 1940, soon after the outbreak of war.

In July, 1940, Yeates joined the Australian Imperial Force and was posted to the 2/9th Battalion (then in England) with the rank of captain and army number U.K. 12. He travelled with the battalion to the Middle East and was the regimental medical officer during the early battles. In 1941 he was posted to the 2/2 Australian Casualty Clearing Station and served with that unit during the siege of Tobruk. During 1942-1943 he served in the South-West Pacific area with the rank of major, working as surgeon in a forward surgical team. During the battle of Buna his surgical post was very near the firing line, and casualties received operative treatment literally on the spot. They were then evacuated by air to Port Moresby. The unit

was situated in a sago-palm swamp, and water used by the unit was obtained by digging with a spade to a depth of 12 inches. As the battle progressed supplies became scarce, and towards the end of the battle conditions became really primitive. Yeates was operating with bare hands, in shorts without a shirt, and for dressings he boiled up those from previous operations and used them wet. He realized that some sleep was necessary, and worked out the minimum number of hours required and gave orders that he was not to be disturbed during that brief period of rest. His senior medical orderlies had been trained by him in resuscitation and were competent to give infusions without supervision, and so on wakening he would find a fresh group of patients all ready for operation. Under these difficult conditions Major Yeates dealt with every type of wound—cerebral, thoracic or abdominal—as it came along. A later follow-up showed that the results of these cases, treated so soon after injury, were better on the whole than those of groups flown out for treatment under better conditions at hospitals in the base area. A medical officer of the Royal Australian Air Force who visited the area during the battle has written:

> I served with the R.A.A.F. and on one occasion was sent over the Owen Stanleys and took the opportunity of spending a night with Jim at his advanced surgical outfit just behind the battle line at Buna. I was absolutely amazed at the work he was doing under incredibly adverse conditions and feel that something should be said about this. . . .

At the end of the battle Major Yeates was mentioned in dispatches for his work. Also his report, written with insight and force, was highly regarded and had some influence on the later conduct of medical operations under such difficult conditions. After being mentioned in dispatches once again for surgical work with the 17th Brigade from Wau to Salamaua, James Yeates was promoted to the rank of Lieutenant-Colonel and appointed O.I.C. Surgical Division, 101 Australian General Hospital.

After his discharge from the Army in 1946, Yeates was appointed honorary assistant surgeon to Sydney Hospital, and visiting surgeon to out-patients, Repatriation Commission. His practice grew steadily, but he found time for many other activities, including tutorship in clinical surgery at the University of Sydney and secretaryship of the Sydney Hospitallers. He served for some years on the council of the Medical Benevolent Society and was vice-president of the Vaucluse branch of the Liberal Party. He produced several short papers on surgical topics derived from a study of the records at Sydney Hospital. These papers are little gems of clinical appraisal, and his generalizations are couched in such attractive and forceful prose that one has not been surprised that they should have often been quoted in journals overseas. During the last few years he became increasingly immersed in a study of diseases of the breast. Owing mainly to his efforts, a special clinic was established at Sydney Hospital, of which he had charge. At the time of his death he was shedding most of his outside interests in order to concentrate on this work as the major effort of his career. Already a great deal of clinical material had been collected, and there is little doubt that with his great gift of literary expression he would have soon been in the forefront in this field.

James Yeates is survived by his widow and two young children. To them and to his mother and brothers we offer our deepest sympathy.

RAYMOND WILLIAM RYAN.

WE are indebted to AIR VICE-MARSHAL E. A. DALEY for the following account of the career of Air Commodore R. W. Ryan.

The death in England on March 30, 1961, of Air Commodore R. W. Ryan (Royal Air Force, retired) brings to a close the life of an Australian who played an important and unique part in the medical services of the Royal Air Force, the Royal Canadian Air Force and the Royal Australian Air Force, and their air crews in the second World War. Born in Victoria on May 26, 1888, he was educated at St. Patrick's College, East Melbourne, and the University of Melbourne where he graduated M.B., B.S., in 1913. Going to England, he served in the Royal Army Medical Corps during the first World War, but with the formation of the Royal Air Force Medical Service in 1919, he obtained a permanent commission with that new force. He took a particular interest in anæsthetics and the medical problems of air crew. His R.A.F. service prior to 1939 covered various posts overseas and in England, but at the outbreak of war he held the important post of President of Medical Boards R.A.F. Central Medical Establishment, London.

In this post, and later as Air Officer Commanding this Establishment, he had a most helpful effect on the R.A.A.F., to the benefit of medical staffs, air crew and others, for through his Establishment all R.A.A.F. in England passed if boards were needed. At the outbreak of war the R.C.A.F. had no Air Force medical service, and the need for this became urgent, particularly with the Empire Air-Training Scheme. As a result Ryan in 1940 was sent on loan to initiate and develop it as the first Director of Medical Services R.C.A.F.; he returned to England in 1943, an R.C.A.F. Air Commodore then took over from him.

Appointed Air Officer Commanding the Central Medical Establishment, he was again in a position to give great assistance to the Dominion Air Forces, which he did with judgement and understanding. He was an amusing personality with a broad knowledge of men, which helped greatly in the difficult problems of disposing of cases on operational service thousands of miles from home. In this the R.A.A.F. medical officers in England and Canada had always a well-informed, knowledgeable and approachable Australian to deal with and to advise them. After the cessation of hostilities, Ryan was appointed to the Health Organization set in post-war Germany, an important and urgent affair. On returning at the end of this duty, he retired, but was made Medical Adviser to the Guild of Air Pilots.

FREDERICK JAMES WILLIAMS.

WE are indebted to DR. V. H. WALLACE for the following account of the career of the late Dr. F. J. Williams.

Frederick James Williams, who died suddenly in his consulting rooms on June 15, 1961, was educated at Wesley College, Melbourne, where I first met him over 50 years ago. Throughout his life Fred was a practical idealist, dedicated to the advancement of human welfare.

As his parents were not well to do, Fred had a struggle to pay for his education in the early days. However, his determination and perseverance helped him to overcome many difficulties, and he finally graduated at the University of Melbourne in 1921. After being a house surgeon at the Victorian Eye and Ear Hospital, Fred became a medical missionary. In this capacity he served in the New Hebrides, China and New Guinea. Subsequently he was a government medical officer in Papua for a period of nine years, first in Samarai and then in Port Moresby. During this period he obtained the diploma of tropical medicine in Sydney.

The last 15 years of Fred's life were spent in active practice as an oculist in Melbourne. He was very happy when he obtained the diploma of ophthalmology. The prevention and treatment of diseases of the eye had greatly appealed to him even when he was a medical student. His special knowledge of this subject obtained for him appointments as ophthalmologist in Fiji and elsewhere. Fred was very popular with patients at the Eye and Ear Hospital. Many people have told me how well he was liked there and how kind he was to everybody. "Children never seemed to cry with him", was one eloquent comment.

The outstanding characteristics in the character of Dr. Fred Williams were sincerity, cheerfulness and courage, and a ready sympathy for the under-privileged. He had a particular abhorrence of war, which he regarded as the supreme folly of mankind. Because of this he was an earnest worker in the pacifist movement. His own church was not particularly interested in pacifism, so quite early in life Fred became a member of the Society of Friends, the Quakers.

As he believed that an international language would help to overcome the barrier to understanding imposed by the diversity of national tongues, Fred was associated with the Esperanto movement for 47 years. He was president of the Australian Esperanto Association for the four years 1956 to 1960. Occupying a permanent place in Fred's consulting room was a portrait of Dr. Zamenhof, the Polish oculist who gave Esperanto to the world in 1887. A memorial service in this international language was held in Melbourne in memory of Dr. Fred Williams.

To his widow and two daughters who survive him we extend our deepest sympathy.

Correspondence.

SHORT-SIGHTED USE OF RESEARCH FUNDS?

SIR: With great respect to the dedicated individuals (academicians, clinicians and laymen) who organize the collection of public funds for research foundations and supervise their application, one might offer an alternative to the widespread policy of drawing only on the interest of some of these funds rather than on the capital.

The community has amply demonstrated its ability and willingness to contribute literally millions of pounds for a worthy cause—for instance, over half a million each for child health and cardiac research. If such funds were used more or less "ad lib"—say at a rate of £50,000 or more *per annum* as required—and the future left to take care of itself, we surely would not be let down by public response in a few years' time to further appeals for replenishing the coffers. But perhaps this is the intention of the trustees of these foundations and other funds anyhow? If not, I hereby put it forward for comment and discussion.

Yours, etc.,

201 Macquarie Street, Sydney.
August 21, 1961.

PETER KENDALL.

THE COMPOSITION OF FLUIDS FOR THE CARE OF PATIENTS WITH ABNORMAL ELECTROLYTE SITUATIONS.

SIR: I note the comments made regarding my paper on solutions for the care of patients with common electrolyte problems, the main point of contention being the line for fluid requirement in anuric adults which for a 70 kg. adult was set at a litre. The paper was written to help general practitioners with hospital patients. It was written as simply as possible with the intention of avoiding complicated issues. Therefore, only brief mention was made regarding anuria, "during anuria maintenance fluid is at once reduced by 50% or more and the patient allowed to lose weight each day. This circumstance is the responsibility of the specialist in the field". The chart, which was constructed in 1955 and has been used at the Royal Children's Hospital since early 1957, does over-estimate the requirements for patients with anuria. The chart was calculated from insensible water loss in anuria from the data of Merrill.[1] This was accepted practice at that time. It became clear, from the comprehensive work of Bluemle, Potter and Elkington,[2] that patients with anuria can obtain about 300 ml. per day from water of oxidation (mainly fat). The insensible water loss in their patients was about 981 ml. $\mp$ 141 ml. The correct amount of maintenance fluid is approximately the sensible water plus insensible water minus water of oxidation and preformed water released from cellular catabolism. This value as found by Bluemle *et alii* is 550 ml. Aware of the assumptions made to predict this amount, they emphasized (page 1105) an increase or decrease depending on circumstances, but warned against using amounts up to 1500 ml. as suggested by Murphy (1952). Other recent workers examining the problem recommend 500 ml. per square metre (Kaplan, Strauss and Yuceoglu)[3] or 860 ml. for an adult during acute renal failure. This fluid is given as intravenous fat emulsion ("Lipomul", 160 Calories per 100 ml.), which promises to supersede the use of aqueous glucose solutions. We have had success with this approach at this Hospital.

Merrill[1] emphasized the variability of fluid requirement in anuria, particularly in the calculation of proper repair of insensible losses, but emphasized the paramount importance of the measurement of the daily weight, which is the determining factor in assessing daily fluid requirement in anuria. There should be a loss of 300 grammes per day in the adult. Merrill's remarks are particularly true in pædiatric patients, where peritoneal dialysis is preferable to the artificial kidney, and where slightly hypertonic fluid is necessary for perfusion to ensure complete return of this peritoneal fluid injected (Segar, Gibson and Rhamy).[4]

Frequently more fluid is returned than is injected. Here the total daily intravenous fluid indicated by weight loss can approximate the amount suggested by the chart.

I did not intend bringing these matters into discussion in this paper, which was not directed at the consultant level. I am in full agreement with the remarks of Ashley and co-workers concerning information at the consultant level and wish to thank them for their comments. "Vehement" disagreement, however, would seem not to be commensurate with the existing situation. A definite amount cannot be struck in this condition, weight must be the guide. One can, however, await the new data of Ashley and co-workers with interest.

Lastly, Dr. Niall has been kind enough to bring forward some valuable clinical observations from his own experience. I agree that it is unwise to restore the sodium concentration completely to normal in water intoxication. In the paper we have indicated the calculation for sodium replacement in severe sodium depletion on the basis of a distribution factor of 0·5 (i.e., total water is assumed to be 50% of the body weight). In water intoxication the total water is said to be 80% of the body weight, so that the use of 0·5 as the distribution factor will not, in fact, return the plasma sodium concentration to normal in water intoxication.

Yours, etc.,

Royal Children's Hospital, Melbourne.
August 22, 1961.

DONALD B. CHEEK.

THE AUSTRALASIAN MEDICAL STUDENTS' ASSOCIATION.

SIR: I was most interested to read your editorial upon the Australasian Medical Students' Association. I note that in this you stated that the British Medical Students' Association was founded in 1942. This is not, in fact, correct, as the Association was founded in 1937 and I was chairman of this in 1939. In 1952 it officially amalgamated with the British Medical Association, and from that time forward enjoyed much help from the parent British Medical Association.

I think that the linking between the two bodies was of the greatest benefit to the Medical Students' Association, and it is to be hoped that a similar close association will develop in this country between the Australasian Medical Students' Association and the Australian Division of the British Medical Association.

Yours, etc.,

52 Elphin Road, Launceston, Tasmania.
August 15, 1961.

S. E. M. BATES.

THE NORTH COAST FUNNEL-WEB SPIDER—ATRAX FORMIDABILIS RAINBOW.

SIR: Recently we had occasion at these Laboratories to investigate the toxicity of female *Atrax formidabilis* venom. It was found to be more toxic on a weight basis than that of female *A. robustus* (Sydney funnel-web spider). A small number of female *A. formidabilis* was obtained by courtesy of Mr. P. Walker of Toowoomba, and we are indebted to him also for the information regarding the habits of these spiders. A specimen was submitted to the National Museum of Victoria, where it was identified by Mr. R. A. Dunn.

Rainbow[1] described the male of this species from a specimen found at Richmond River, New South Wales. Musgrave[2] and Ingram and Musgrave[3] reported a case of a bite by a male *A. formidabilis* in a strong and healthy sleeper-cutter who, after becoming desperately ill a few hours after the bite, eventually recovered. The clinical picture of envenomation resembled closely that observed in persons bitten by male *A. robustus*. Ever since, the spider has been considered as dangerous to humans.

The approximate LD_{50} for female *A. formidabilis* venom as determined in 15-17 gramme albino mice is: 0·2 mg. intravenously, 0·35 mg. intraperitoneally and 0·45 mg.

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[1] "The Treatment of Renal Failure", 1955, Grune & Stratton, New York and London: 83.

[2] *J. clin. Invest.*, 1956, 35: 1094.

[3] *Pediatrics*, 1960, 25: 409.

[4] *Pediatrics*, 1961, 27: 603.

[1] *Records of the Australian Museum*, 1914, 10: 255.

[2] *Records of the Australian Museum*, 1927, 16: 33.

[3] MED. J. AUST., 1933, 2: 10.

subcutaneously. The picture of envenomation in mice closely resembles that observed with *A. robustus* venom as described earlier by Wiener,[4] and it is evident that a bite by a female *A. formidabilis* is more dangerous to mice, as the LD_{50} indicates a higher toxicity.

The average venom yields during the first four milkings (at weekly intervals) of a number of spiders kept at these Laboratories, was 0·55 mg. of dry venom per spider.

When paper-strip electrophoresis of the venom was carried out, the pattern obtained was different from those obtained with male and female *A. robustus* venoms.

McKeown[5] states that males of this species live in hollow trees. Mr. P. Walker, who has been observing the habits of these spiders for several years, has informed us that female *A. formidabilis* are found in burrows in the soil, which may reach a depth of two feet. The interior of the burrow is lined with a free-hanging dense web. The burrow has two enlargements, the upper one for the egg sack, the lower (usually about eight inches deeper) for the spiderlings, after hatching. Mr. Walker has never found a male in such a burrow.

On the basis of our observations, we conclude that it is possible that even a bite of a female *A. formidabilis* may produce ill effects in humans. We expect to extend our observations to the male spiders of the species.

Yours, etc.,

Commonwealth Serum Laboratories, G. H. KAIRE.
Parkville,
Victoria.
August 21, 1961.

A COMPARISON OF THE NEWER DIURETICS, TRICHLORMETHIAZIDE, BENDROFLUAZIDE AND CHLORTHALIDONE WITH CHLOROTHIAZIDE.

SIR: I agree with Dr. J. E. Gault (MED. J. AUST., August 26, 1961) that the cost of two equally effective drugs should be considered in our prescribing, and that in this case the cheaper one should be used. However, the Pharmaceutical Benefits Scheme, with its 5s. flat-rate method of payment, gives no incentive whatsoever to consider cost.

It is, of course, a fact that there are effective corticosteroids not included in the Government list which are cheaper than those in the list. There are many treatments outside the list cheaper than many that are in the list. Further, within the list itself, no incentive is given to use the less expensive preparations—they cost 5s. irrespective, and even if one orders less than the maximum quantity the patient is not offered any reduction.

These wasteful conditions would cease to exist if the patient paid some small fraction of the cost instead of a flat rate, and most of the regulations and restrictions which form an inevitable part of a flat-rate scheme would be unnecessary, if the cost to the patient were related to the actual cost of the prescription.

Yours, etc.,

267 Bobbin Head Road, D. J. MARR.
North Turramurra,
New South Wales.
August 25, 1961.

PROBLEMS OF CANCER DETECTION.

SIR: In your publication of July 29 were four papers on cancer detection of the cervix uteri. The overall picture given is very broad, and Ian Cope made a very good case for the use of the colposcope in detecting cervices requiring biopsy.

Howell, in his paper, draws attention to the alteration of many abnormal smears to normal after injecting "Œstrogenine". Howell points out that in the mass production of smears there are many side issues where the patient may be lost sight of, or where one may produce inaccurate diagnosis, or even cases of cancer may be missed in the early stages.

A great deal depends upon the team. When with Joe Meigs and Ruth Graham, one was obsessed with the 100% accuracy of the Papanicolaou smear, whereas Te Linde, at Johns Hopkins, was less sanguine. Papanicolaou himself prefers the smear taken from the vaginal pool, because here are found most of the desquamated cells. The smear taken by Ayre's spatula may miss the actual area where early carcinoma is developing. This may be made more accurately when combined with colposcopy.

Cope showed the value of colposcopy in his reference to Navratil, who found 9% of carcinoma amongst the abnormal colposcopic findings.

In taking smears there should be two smears from each case, one from the vaginal pool and one by Ayre's spatula, or by a cotton-wool-dressed probe taking the smear from the endocervix. These two smears give the pathologist an increased capacity for more accurate assessment of abnormal cells and the indication of malignancy or otherwise to the clinician.

Yours, etc.,

63 Palmer Place, R. F. MATTERS.
North Adelaide.
August 28, 1961.

THE PRINCIPLES OF SURGICAL NURSING.

SIR: Like many an author before me, I suppose, I was a little disappointed that my book, "The Principles of Surgical Nursing", did not attract more favourable notice from your reviewer. I do not complain. Indeed, I am grateful for the tribute to my collaborators and for your reviewer's undertaking to recommend the book to his ward sister if I will change one word in the title.

It may perhaps amuse your readers to see another review, unpublished, which was sent to me privately soon after the book came out. The writer, who wishes to remain anonymous, has given me permission to transcribe it in this letter.

Just everything the surgeon knows
Has been distilled by Thomas Rose
Into a mere six hundred pages,
In which the wisdom of the ages
Is brought in language clear and terse
To educate the willing nurse.

The nurse may now give up conjectures
And likewise cut untimely lectures.
This inexpensive mental fattener
Can be her passport to the ATNA.
It is, as Matron said to me,
The Bible of the N.R.B.

I admit that this overpraises the work as much as (I would like to think) your reviewer underpraises it.

Yours, etc.,

141 Macquarie Street, T. ROSE.
Sydney.
August 19, 1961.

GLUTEN-FREE DIETS IN AUSTRALIA.

SIR: We were very interested to read Dr. Theo Cooke's article in the August 19 issue on "Gluten-Free Diets in Australia", with its very helpful compilation of foods which do contain wheat gluten. However, we would like to make some comments.

Dr. Cooke infers that the exclusion of gluten-containing wheat flour reduces the intake of protein and calcium below the recommended levels. This should not be so if the diet is administered correctly. The gluten-free diet as recommended by the Royal Children's Hospital, Melbourne, is based on the concept of protective foods, e.g., milk, eggs, meat or fish, fresh fruit, vegetables, butter and fortified margarine, and a very adequate protein and calcium intake will be obtained if correct use of these is made. There should then be no need for the addition of expensive foods such as "Complan" and "Nesmida".

We do feel that Dr. Cooke has based many of his statements regarding the diet on English, not Australian, food habits. His method of presenting the diet is somewhat complex, and he states that it can be monotonous, unpleasant and restricted. This need not be so. It has been our practice to stress the simplicity of the diet and its approximation to normal, apart from the exclusion of wheat and rye gluten-containing foods. Monotony is avoided by education of the parent or patient, if adult, in the proper

[4] MED. J. AUST., 1957, 2: 377.
[5] "Australian Spiders", 1952.

use of foods and substitutes, so that their meals are as similar to the family meal as possible. In this way also, shortages of any essential vitamins or foodstuffs are avoided.

The recipe as printed for gluten-free bread is incorrect, but even if this bread were made by the correct recipe it could not be described as having a normal bread taste and texture. However, it is a very palatable and useful product. The recipe containing glucono-delta-lactone was first worked out by Townsend and Rowe and tested with our cooperation and comment, and the correct recipe is recorded in *The Journal of the Dietetic Association of Victoria*, September, 1960, Volume 2, page 7, and in the booklet "Diet in Cœliac Disease" compiled by the Dietary Department and the Clinical Research Unit, Royal Children's Hospital, Melbourne. The latter also incorporates many recipes. The bread mix available from Sanitarium Health Foods is prepared according to the correct recipe using glucono-delta-lactone.

Finally, perhaps a word should be said about the availability of cornflour or special wheat starch. It is now the practice of the majority of commercial firms to market wheat starch as cornflour, with the statement on the packet that the product is made from wheat. This has been done because of the ready availability and cheapness of wheat starch compared with true cornflour. Therefore it will be very difficult for patients to obtain real cornflour, especially as recently Parsons' "Globe" brand has been withdrawn from the market. As far as we know, Brown and Polsons' cornflour is the only genuine maize product in Victoria. However, we do not consider that the amount of gluten remaining in the "wheat starch" cornflours is significant, as in our experience in the laboratory it does not rise above 0·1% to 0·3%. It is probably wishful thinking to imagine that one can achieve a diet which contains less gluten than this, and in our clinical experience it does not matter in the vast majority of cases.

We believe strongly that it is important to achieve simplicity with this diet, so that it will fit into the normal meal pattern in the home. Otherwise it is not likely that patients will keep to the diet during the long period that is necessary.

Yours, etc.,

CHARLOTTE M. ANDERSON, M.D., M.Sc.,
Deputy Director, Clinical Research Unit.
SHEILA M. SUTHERLAND,
Chief Dietitian.
Royal Children's Hospital,
Melbourne.
August 28, 1961.

AUTONOMIC DYSPRAXIA AND DANDRUFF.

SIR: I would be obliged if I might use your columns to correct some details reported in today's *Sun-Herald* concerning the relationship of anxiety and dandruff. A very welcome, controlled series carried out by Mr. B. Fenelon, psychologist at Newcastle University College, is described. In tests with 40 university students, he found that as they became more nervous, the amount of dandruff in their hair increased.

This is a most interesting piece of work. I am looking forward to studying Mr. Fenelon's paper, as soon as it becomes available. The article states that a connexion between nervousness and dandruff has been discovered by Mr. Fenelon; "confirmed" would appear to be a more appropriate verb, in view of the fact that the association was reported by me in "Autonomic Dyspraxia" (1958).[1]

The second point is the statement: "There is no direct physiological mechanism which could cause this to happen." The hypothesis of autonomic dyspraxia offers a physiological, or more accurately, a pathological, mechanism explaining how dandruff can be influenced by such remote factors as anxiety—how it can be caused by anxiety.

The hypothesis proposes that emotional tension reacts on the hypothalmus, the autonomic brain, resulting in commotio hypothalami, which, in turn, interferes with the central coordination of the peripheral autonomic nervous system. The function of the peripheral autonomic nervous system is essentially the control of the cellular activity of the peripheral organs. In this case the peripheral organ is the skin of the scalp, whose cells function according to the neurocirculatory and neurohormonal activity of the autonomic nervous system. This, in the final analysis, is controlled by the central autonomic nervous system, loosely termed the hypothalmus: more accurately, the autonomic brain. In health and happiness central autonomic homœostasis results in peripheral physiological activity. In emotional tension homœostasis is replaced by commotio hypothalami, which causes peripheral pathology.

Whilst this mechanism is obviously simple in the case of a person fainting at the scene of an accident, where the time factor between the cause and effect is unmistakable, it is not so obvious in the case of an organ as obscure as the skin, and in a condition where the emotional stress is of a lesser magnitude and the time factor indefinite. But the same factors apply in every organ. The basal cells of the skin must mature at the correct *tempo*, and a perfect homœostasis of the glands and other structures of the scalp must exist for a healthy scalp. If this delicate balance fails through a failure of central coordination, the local conditions are favourable for a multiplication of the micro-organisms which "produce" dandruff.

The relation of another function of the skin to emotional stress is well established: sweating. Psychologists are well acquainted with it. It is the basis of the psycho-galvanometer reaction. It becomes as chronic a pathological condition as dandruff in anxiety neurosis. The mechanism again is explained by the hypothesis.

I was particularly pleased to read of Mr. Fenelon's confirmatory work in view of Russell Cecil's scepticism on this point. Although he made no mention of it in the foreword he so kindly wrote for "Autonomic Dyspraxia", he mentioned in subsequent correspondence that dandruff was the one condition of the many listed in which he doubted that emotional tension was concerned.

Yours, etc.,

BRIAN HAYNES.
"Craignish",
185 Macquarie Street,
Sydney,
New South Wales.
August 27, 1961.

[1] "Autonomic Dyspraxia", 1958, Lewis, London and New York.

PHENYLKETONURIA.

SIR: With reference to the leading article on "Phenylketonuria" in the August 26, 1961, issue of THE MEDICAL JOURNAL OF AUSTRALIA, I wish to point out that routine urine testing for phenylketonuria has been carried out in the Maternal and Child Welfare Centres throughout Queensland since January 22, 1960.

Yours, etc.,

H. C. MURPHY,
Director.
Maternal and Child Welfare Centres,
184 St. Paul's Terrace,
Brisbane.
August 29, 1961.

THE USE OF UNMODIFIED BLOOD GIVEN BY DIRECT TRANSFUSION.

SIR: Although Dr. Dique states (MED. J. AUST., August 19, 1961) that there are many non-urgent cases in which the use of unmodified blood is undoubtedly better than that which has been stored, I think that direct transfusion is even more beneficial in urgent cases of hæmorhage due to blood dyscrasia. Recently at the Alfred Hospital, a boy, aged 14, had severe hæmorrhage after a heart operation. I was urgently called to give a direct transfusion, and using five donors consecutively within a period of one hour, bleeding was controlled.

With regard to cross matching, I do not think that more care is necessary in direct transfusions. There is less likelihood of giving the wrong blood, as the donor knows his group and carries a Red Cross card with him. On arrival at the Blood Bank, the donor's name and group are checked from his card. The serologist takes blood by skin puncture into Rous and Turner solution. The compatibility test is set up in saline and albumin respectively, and the albumin mixture is incubated at 37° C. After 10 minutes' incubation a preliminary inspection is made by myself, and after one hour the final reading by the serologist. In cases where only one donor is used, if the test is satisfactory, I proceed with the transfusion. However, when two or more donors are used, or an indirect Coombs test

is required, the compatibility tests are performed on the day previous to the transfusion.

Any untoward reaction in the patient is noticed immediately it occurs during the transfusion and remedial treatment is given at once. Such reactions, although rare, may be due to antibodies not recognized by routine cross matching.

Yours, etc.,

JOHN McLEAN.

417 St. Kilda Road,
Melbourne.
August 29, 1961.

British Medical Association.

MERVYN ARCHDALL MEDICAL MONOGRAPH FUND.

THE following is a list of donations received for the Mervyn Archdall Medical Monograph Fund:

	£	s.	d.
Professor Lorimer Dods	21	0	0
Dr. D. O. Crompton (North Adelaide), Dr. R. H. Macdonald (Sydney) ..	10	10	0
Dr. Bryan Gandevia (Melbourne), Dr. E. Kent Hughes (Armidale), Dr. K. S. Jones (Sydney)	5	5	0
Total	£57	15	0

Medical Practice.

NATIONAL HEALTH ACT, 1953-1961.

THE following announcement is published in the *Commonwealth of Australia Gazette*, No. 59, of July 21, 1961.

NATIONAL HEALTH ACT 1953-1961.

Notice in Pursuance of Section 134A.

Notice is hereby given that following investigation and report by the Medical Services Committee of Inquiry for the State of New South Wales concerning the conduct of Margers Bertolds Baltgailis, of Rooty Hill-road, Rooty Hill, medical practitioner, I, Donald Alastair Cameron, Minister of State for Health, did on the eleventh day of July, 1961, reprimand the said Margers Bertolds Baltgailis for conduct which is a breach of the National Health Pharmaceutical Benefits) Regulations.

Dated this eleventh day of July, 1961.

DONALD A. CAMERON,
Minister of State for Health.

The following announcements are published in the *Commonwealth of Australia Gazette*, No. 62, of August 3, 1961.

NATIONAL HEALTH ACT 1953-1961.

Notice in Pursuance of Section 134A.

Notice is hereby given that the Medical Services Committee of Inquiry for the State of New South Wales, after investigation, having reported on the thirtieth day of March, 1961, concerning the conduct of Kevin Joseph Fitzgerald, of Forest-road, Peakhurst, a medical practitioner, in relation to his provision of medical services under Part IV. of the *National Health Act* 1953-1961, I, Donald Alastair Cameron, Minister of State for Health, did on the eleventh day of July, 1961, reprimand the said Kevin Joseph Fitzgerald.

Notice is hereby given that following investigation and report by the Medical Services Committee of Inquiry for the State of New South Wales, concerning the conduct of Sydney Peter Addison Henderson of Vardys-road, Seven Hills, medical practitioner, I, Donald Alastair Cameron, Minister of State for Health, did on the eleventh day of July, 1961, reprimand the said Sydney Peter Addison Henderson for conduct which is a breach of the National Health (Pharmaceutical Benefits) Regulations.

Dated this eleventh day of July, 1961.

DONALD A. CAMERON,
Minister of State for Health.

The following announcement is published in the *Commonwealth of Australia Gazette*, No. 64, of August 10, 1961.

NATIONAL HEALTH ACT 1953-1961.

Notice under Section 134A.

I, Donald Alastair Cameron, the Minister of State for Health, in pursuance of the powers conferred by section 134A of the *National Health Act* 1953-1961, hereby give notice that I have revoked the authority of Reginald John Nowland, of 69 Warringah-road, Narraweena, in the State of New South Wales, medical practitioner, under section 88 of the said act to write a prescription for the supply of pharmaceutical benefits and under section 93 of the said act to supply pharmaceutical benefits, in pursuance of my powers under sub-section (2.) (a) of section 133 of the said act.

Dated this twenty-first day of July, 1961.

DONALD A. CAMERON,
Minister of State for Health.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 62, of August 3, 1961.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Termination of Appointments.—The appointments of Robert Charles Mulholland and Kenneth Nicholson Howson as Surgeon Lieutenants (for Short Service) are terminated, dated 1st July, 1961, and 17th July, 1961, respectively.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Promotions.—Surgeon Lieutenant Bryan Hudson is promoted to the rank of Surgeon Lieutenant-Commander, dated 15th March, 1961.

The following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 64, of August 10, 1961.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Extension of Retiring Age.—The retiring age of Surgeon Rear-Admiral Lionel Lockwood, C.B.E., M.V.O., D.S.C., is extended for a period of two years from 13th January, 1962.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Appointments.—Lieutenant Mervyn John Richards, Royal Australian Naval Reserve, is appointed Surgeon Lieutenant (D), with seniority in rank of 31st January, 1950, dated 5th July, 1961.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).—To be Temporary Majors—Captains 48053 T. T. Teusner, 10th July, 1961, and 240222 P. N. McGuire, 29th July, 1961.

Citizen Military Forces.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—To be Captain (provisionally), 26th June, 1961—2146649 Brian Michael Dwyer.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointment of 3129455 Captain D. R. Macdonald is terminated, 6th January, 1961.

Central Command.

Royal Australian Army Medical Corps (Medical).—432064 Major R. S. Colton is appointed from the Reserve of Officers, 27th June, 1961.

Northern Territory Command.

Royal Australian Army Medical Corps (Medical).—73018 Captain I. D. Byrne is appointed from the Reserve of Officers and to be Temporary Major, 23rd January, 1961.

Reserve Citizen Military Forces.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—To be Honorary Captain, 4th July, 1961—Alexander Douglas.

Southern Command.

Royal Australian Army Medical Corps (Medical).—To be Honorary Captain, 7th January, 1961—Donald Ross Macdonald.

Notes and News.

Precautions against Cholera.

The Minister for Health, Dr. D. A. Cameron, has announced that as a result of outbreaks of cholera in Hong Kong and Macao, the necessary steps have been taken to require vaccination certificates against cholera for passengers in ships or aircraft arriving from these areas. Such arrangements are already in force in relation to passengers arriving in ships and aircraft from India and Pakistan. Shipping and aircraft companies operating from these areas have been requested to warn all intending passengers of this quarantine requirement.

Supplies of Salk Vaccine.

The Minister for Health, Dr. D. A. Cameron, has announced that about 160,000 doses of Australian-made Salk poliomyelitis vaccine are expected to be ready for distribution before the end of this month. This supply is in addition to the batch of 600,000 doses which are now undergoing final tests at the Commonwealth Serum Laboratories. The 160,000 doses that have now become available comprise vaccine from earlier batches which had been delayed by testing difficulties, but have now passed all tests satisfactorily. Part of this material will require to be subjected to final sterility tests after it has been bottled, but there is no reason to expect that its delivery will be delayed beyond the end of the month. Dr. Cameron states that the Special Committee he asked to examine production and testing problems at the Commonwealth Serum Laboratories have reported to him that these difficulties appear to have been satisfactorily overcome, and it is unlikely that it will need to meet again.

World Rehabilitation Fund Fellowships.

The World Rehabilitation Fund has a limited number of fellowships available for physicians from other nations for post-graduate training in physical medicine and rehabilitation in the United States. The fellowships are for a one year period, but are renewable to a maximum of three years by mutual consent of the awardee and the World Rehabilitation Fund. Training under the fellowships is given in the Department of Physical Medicine and Rehabilitation, New York University Medical Center, of which Howard A. Rusk, M.D., is chairman.

Recipients of fellowships receive a stipend of $3400 per year, when room and board are not furnished, and a stipend of from $1900 to $2900 per year when room and board are provided. For each dependant, up to three, accompanying the awardee to New York, there is an additional allotment of $25 per month. There is one increase of $300 after the first year.

Visa for awardees are sponsored by the New York University Exchange Visitors Program. The fellowships do not supply transportation to and from the awardee's country of origin; transportation is the responsibility of the awardee.

Awardees are required to have had their credentials evaluated by the Educational Council for Foreign Medical Graduates, and must have successfully passed the examination of the Educational Council for Foreign Medical Graduates prior to applying for a fellowship. In addition, one year's internship is required. The internship may be fulfilled in the United States or in the awardee's nation. When the selection of the awardee is made, it will be contingent upon the evaluation of his credentials and successful completion of the examination of the Educational Council for Foreign Medical Graduates.

Awardees should plan to begin their training on July 1 or January 1.

In addition to the professional training programme, the awardee should correspond directly with Peter Stratis, M.D., Director of Professional Education, Department of Physical Medicine and Rehabilitation, New York University Medical Center, 400 East 34th Street, New York 16, New York.

An Honour for Dr. K. L. H. Kirkland.

At the twelfth Congress of the International Society of Urology, held at Rio de Janeiro, Brazil, in July, 1961, Dr. K. L. H. Kirkland was elected a Vice-President of the Society.

Third International Congress of the Pathology of Infectious Diseases.

The International Society for the Study of Infective and Parasitic Diseases announces that the third International Congress of the Pathology of Infectious Diseases will be held in September, 1961, at Bucharest, under the presidency of Professor S. Nicolau. The scientific programme will comprise a round-table discussion and three symposia. The subject of the round-table discussion will be "Enteropathies of Viral Origin", and the speakers will be J. D. Verlinde (Leiden), A. Giovanardi (Milan) and M. Pinto (Lisbon). The symposia will be as follows: (i) "Infectious Mononucleosis", chairman R. Sohier (Lyon); (ii) "The Immunizations of Childhood", chairman G. Fanconi (Zurich); (iii) "Steroid Therapy of Acute Infectious Diseases", chairman L. Heilmeyer (Freiburg). Further information may be obtained from the Secretary-General, Professor R. De Mattia, at the following address: Società Internazionale per lo Studio delle Malattie Infettive e Parassitarie, Ospedale "Amadeo di Savoia", Corso Svizzera, 164, Turin, Italy.

Services Canteens Trust Fund.

Post-Graduate Scholarship.

The trustees of the Services Canteens Trust Fund are inviting applications for a post-graduate scholarship for study overseas. The fields of study in which the scholarship may be awarded are: (i) any course at any approved university throughout the world; (ii) aeronautics in England or America; (iii) travelling scholarship in any field. An applicant wishing to pursue any other branch of study may apply to the trustees for a scholarship in that field.

The scholarship is valued at £A1000 *per annum*, and will be tenable for a period of up to three years.

The scholarship is open to a child (including step-child, adopted child or ex-nuptial child) of a person who was at any time between September 3, 1939, and June 30, 1947, (*a*) a member of the Naval, Military or Air Forces of the Commonwealth, or (*b*) a member of any nursing service or women's service attached or auxiliary to any branch of the Defence Force of the Commonwealth, or (*c*) a member of the canteens staff of any ship of the Royal Australian Navy, and any person duly accredited to any part of the Defence Force who served in an official capacity on full-time paid duty.

Selection will be entirely on merit, and will be competitive. The scholarship will be granted only to an applicant who, in the opinion of the trustees, has outstanding ability, is of suitable character and is likely to obtain lasting benefit to himself or herself and to Australia by further study. The scholarship will not necessarily be awarded each year.

The following will be taken into consideration in determining the award of the scholarship: (i) academic career; (ii) ability for research work; (iii) character; (iv) the future value to Australia of the subject of research selected. The selection each year of the scholar to be awarded the scholarship will be made from all applications received from eligible persons by the prescribed closing date.

Applications must be lodged with the General Secretary, Services Canteens Trust Fund, Victoria Barracks, St. Kilda Road, Melbourne, by November 1, 1961. Applications should be transmitted through the Regional Secretary in the State in which the applicant resides.

Application forms and any further information may be obtained from the General Secretary, whose address is shown above, or from a Regional Secretary of the Fund. The addresses of Regional Secretaries are as follows: Queensland, Victoria Barracks, Brisbane; New South Wales, 84 Pitt Street, Sydney; Victoria, Victoria Barracks, Melbourne; South Australia, 22 Grenfell Street, Adelaide; Western Australia, Swan Barracks, Perth; Tasmania, Anglesea Barracks, Hobart.

The Health Service in Jugoslavia.

The following information relating to the health service in Jugoslavia appears in *La Presse médicale* of June 17, 1961.

The new law governing the organization of the health service in Jugoslavia stipulates that those who insure themselves with the social security fund (almost the whole population of the country) must have free choice of doctor. The insured person may choose a medical institution (medical clinic, a general clinic, a dispensary, an ambulance or a health station) and one of the doctors attached to this establishment; he must then enter into a contract with the institution of his choice of one year's duration, during which period he will be cared for by his chosen doctor and at the expense of the institution; the costs (examination, medicines, laboratory and radiological investigations) are repaid by the social security fund. If the patient is not satisfied at the end of a year, he may freely choose another institution where he will be treated.

According to the new law, every medical institution must carry out its work in exchange for the payments that it receives from the social security fund for services rendered to insured patients.

The salaries of the doctors and all health service personnel are partly dependent on the "variable surplus" in accordance with the amount of work carried out; that is to say, the more patients the institution has, the greater will be the payments received and the higher the salaries of the personnel, including the doctors. Each medical institution must have, besides a director, a board of management consisting of several members of the health staff of the establishment and several members (the number depending on the importance of the institution) of other social groups or even private citizens (social management).

Honours.

1958 AUSTRALIAN NATIONAL ANTARCTIC RESEARCH EXPEDITIONS.

HER MAJESTY THE QUEEN has been graciously pleased to award the Polar Medal to Dr. James Edward Grey Channon for services rendered during the 1958 Australian National Antarctic Research Expeditions.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course in Cardio-Vascular Diseases.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in recent advances in cardio-vascular diseases will be held in the

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 12, 1961.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	..	2	..	..	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	5	..	..	..	2	..	7
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1(1)	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	3	5(4)	2(2)	..	3	..	8	1	22
Diphtheria	1	1(1)	..	..	..	..	..	..	2
Dysentery (Bacillary)	..	8	..	..	5(3)	..	2	..	15
Encephalitis	1(1)	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	2	..	..	2
Infective Hepatitis	87(20)	72(26)	14(4)	23(11)	2(1)	..	2	7	207
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	2	..	..	..	..	..	2
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	..	..	..	1	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(2)	..	5(5)	3(2)	..	..	..	..	13
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	25(7)	..	3(2)	1(1)	..	..	..	29
Salmonella Infection	..	..	..	1(1)	..	..	..	..	1
Scarlet Fever	10(6)	17(6)	2(2)	9(6)	..	..	..	1	39
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	30(19)	23(20)	20(9)	1(1)	7(6)	3	..	..	84
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Maitland Lecture Hall, Sydney Hospital, from 10 a.m. to 5 p.m. on Saturday, October 7, and from 10 a.m. to 12.30 p.m. on Sunday, October 8, 1961. The fee for attendance is £3 3s. Early application, enclosing remittance, should be made to the Course Secretary, The Post-Graduate Committee in Medicine, Herford House, 188 Oxford Street, Paddington. Telephone: FA 0671.

SEMINARS AT THE ROYAL PRINCE ALFRED HOSPITAL.

Alterations to the Programme.

THE following alterations in the programme of seminars at the Royal Prince Alfred Hospital for the second half of 1961 have become necessary.

September 8, previously Thoracic Section, now Psychiatric Section, subject to be announced, Dr. Murray Jackson, Consulting Psychiatrist, Guy's Hospital, London (by invitation). September 22, previously Pædiatric Section, now Thoracic Section, "Chronic Cor Pulmonale", Professor K. Donald, Edinburgh (by invitation).

October 13, previously Psychiatric Section, now Thoracic Section, "Some Problems of Lung Cancer", Dr. H. M. Rennie, Mr. Rowan Nicks.

November 3, previously Thoracic Section, now Pædiatric Section, "Anæmia in Infancy and Childhood", Dr. M. Harris.

The Thoracic Section seminar on pulmonary embolism has been postponed until 1962.

Notice.

THE AUSTRALASIAN ASSOCIATION OF PSYCHIATRISTS (QUEENSLAND BRANCH).

Guest Speaker.

DR. D. MADDISON, Professor-Elect in Psychiatry, University of Sydney, will be a guest speaker for the Australasian Association of Psychiatrists (Queensland Branch) in Brisbane on the following dates. He will address a clinical meeting at Lowson House, Brisbane General Hospital, at 2 p.m. on September 29, 1961, and will give an open lecture at 8 p.m. in the Edwin Tooth Memorial Lecture Room on the subject "Pattern of Illness in Families". A clinical meeting will be held at the Brisbane Mental Hospital, Goodna, at 1.30 p.m., on September 30, 1961.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

McLachlan, John, M.B., B.S., 1959 (Univ. Sydney), c.o. Public Health Department, Kavieng, New Ireland, Territory of Papua and New Guinea.

Nathaniel, Rajah Lionel, M.B., B.S., 1960 (Univ. Sydney), Canterbury District Memorial Hospital, Canterbury Road, Campsie.

Deaths.

THE following deaths have been announced:

LEMMON.—William Morton Lemmon, on August 24, 1961, at Melbourne, Victoria.

AHERN.—Albert John William Ahern, on August 26, 1961, at Melbourne.

BEITH.—Bruce McNeil Beith, on August 27, 1961, at Uralla, New South Wales.

LAMBIE.—Charles George Lambie, on August 28, 1961, at Sydney, N.S.W.

Diary for the Month.

SEPTEMBER 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPTEMBER 14.—New South Wales Branch, B.M.A.: Public Relations Committee.
SEPTEMBER 15.—New South Wales Branch, B.M.A.: Ethics Committee.
SEPTEMBER 18.—Victorian Branch, B.M.A.: Finance Sub-Committee.
SEPTEMBER 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPTEMBER 20.—Western Australian Branch, B.M.A.: General Meeting.
SEPTEMBER 20.—Victorian Branch, B.M.A.: Clinical Meeting (Victorian Eye and Ear Hospital).
SEPTEMBER 21.—Victorian Branch, B.M.A.: Executive Meeting of Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): Medical Officers to Sydney City Council. All contract practice appointments in New South Wales. Members are requested to consult the Medical Secretary before undertaking practice in dwellings owned by the Housing Commission.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full data in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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